# THE PHYSIOLOGY AND PATHOLOGY OF THE CEREBRAL CIRCULATION

LEONARD HILL

THE

# PHYSIOLOGY AND PATHOLOGY

OF

# THE CEREBRAL CIRCULATION

## AN EXPERIMENTAL RESEARCH

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# PREFACE.

In the following pages will be found the substance of three Lectures which I delivered as Hunterian Professor at the Royal College of Surgeons in February last.

I have largely been enabled to carry out the researches of the past five years therein detailed, through the munificence of the Worshipful Company of Grocers. This volume contains a report of the work done by me as their Research Scholar. In writing some of the sections of this work I have drawn on papers previously published by me in the *Proceedings of the Royal Society*, the *Journal of Physiology*, and the *British Medical Journal;* other sections, such as those on the Cerebral Pulsation, the Cerebro-spinal Fluid, Cerebral Anæmia, and Cerebral Compression are new, and contain an account of experiments hitherto unpublished.

I have been partly supplied with the means of research by grants from the Royal Society Government Grant, and the British Medical Association; and I owe a debt of gratitude to Professor Burdon Sanderson, Professor Schäfer and the Authorities of the London Hospital Medical College, whose laboratories and laboratory appliances have been placed at my disposal.

I have gained very greatly from the criticisms of my friends, Professor Burdon Sanderson, Professor Schäfer, Dr. Rose Bradford, Mr. B. Moore, Dr. Arthur Keith, Dr. J. H. Sequeira, Mr. Harold Barnard, Mr. C. Wall and many Members of the Physiological Society, who have at different times watched my experiments and corroborated my results.

I am deeply indebted to Mr. Bayliss and Mr. Nabarro, who have been my co-workers in part of these researches.

Mr. Langley has been generous enough to lend me, from the *Journal of Physiology*, Figures 5, 6, 7, 8, 11, 13, 14, 15, 16, 17, 18, 19, 20, 21; for Figures 35, 36, 37 I am indebted to the publishers of Morris's "Anatomy." To all these friends I tender my warmest thanks.

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# CONTENTS.


PAGE

INTRODUCTION:

Origin and Plan of Research . . . . . 1

SECTION I.—PULSATIONS OF THE BRAIN.

History . . . . . 5
Method of Research—New Instruments . . . . . 8
Cerebral Pressure Gauge . . . . . 9
Pulsation of Cerebro-spinal Fluid . . . . . 10
The Brain Pulsates in the Closed Cranium . . . . 10
Pulsation of Cerebral Veins . . . . . 10
Cerebral Venous Circulation—Method of Investigation . . 11
Resemblance of the Cranial Cavity to an Oncometer . . 12
Fallacies of the Oncometric Method . . . . . 12
Absence of Valves in the Cerebral Veins . . . . . 13
Pathological Changes in the Cerebral Pulsation . . . 13
SUMMARY OF SECTION I. . . . . 15

SECTION II.—THE CEREBRO-SPINAL FLUID.

History . . . . . 16
Anatomy of the Subdural and Subarachnoid Spaces . . . 16
EXPERIMENTS ON THE INJECTION OF FLUID INTO THE CRANIO-VERTEBRAL CAVITY . . . . . 18
History . . . . . 18
Method of Research . . . . . 19
Irrigation of the whole Cranio-vertebral Cavity . . . 19
Filtration of Fluid from the Cranial Cavity . . . . 20
The Pressure at which Filtration takes place . . . . 20
Filtration through the Dura Mater . . . . . 20
A Typical Experiment on the Filtration of Fluid . . . 21

PAGE

The Pathways of Filtration . . . . . 22
The Secretion and Absorption of Cerebro-spinal Fluid . 23
Amount of Cerebro-spinal Fluid . . . . . 24
Subarachnoid Space a Potential but not Actual Space . . 25
Transudation of Cerebro-spinal Fluid, its Dependence on Cerebral Venous Pressure . . . . . 27
Summary of Section II. . . . . 28

## Section III.—THE CEREBRAL CIRCULATION.

The Monro-Kellie Doctrine . . . . . 30
History . . . . . 31
Fallacies of Post-mortem Examination . . . . . 33
The Monro-Kellie Doctrine tested by Experiment . . . 35
History of Previous Experiments . . . . . 35
Method of Research . . . . . 36
History of Previous Methods of Investigating the Cerebral Circulation . . . . . 37
Method of Research . . . . . 40
Search for Cerebral Vaso-motor Nerves . . . . 42
Method of Experiment . . . . . 42
Stimulation of Vago-sympathetic Nerves . . . . 43
Stimulation of Dorsal Spinal Cord . . . . . 43
Stimulation of Stellate Ganglion . . . . . 45
Histological Search for Cerebral Vaso-motor Nerves . . 45
Previous Researches into Cerebral Vaso-motor Nerves . . 45
Negative Results . . . . . 46
Effect of Drawing a Ligature round the Neck . . . 46
Stimulation of the Peripheral End of the Vagus Nerve . 46
Discussion of some Physical Conditions of the Cerebral Circulation . . . . . 46
Direct Continuity of the Cerebral Veins with the Thoracic Veins . . . . . 49
Absence of Valves . . . . . 49
Cerebral Venous Pressure varies absolutely as Pressure in the Venæ Cavæ . . . . . 50
Intracranial Pressure is the same as Cerebral Venous Pressure . 50
Intracranial and Cerebral Venous Pressure vary directly but not absolutely as Arterial Pressure . . . . . 50

PAGE

Cerebro-spinal Fluid Pressure fails to vary absolutely as Intracranial Pressure . . . . . 50
A Typical Experiment . . . . . 50
Confirmation of Experiments by Previous Workers . . . 51
The Cerebral Vessels as Rigid Tubes . . . . . 51
Effect of Blocking Cerebral Veins during Excitation of Vagus . 52
Discussion of Previous Results . . . . . 54
Excitation of Sensory Nerves, Effect on the Cerebral Circulation . . . . . 55
Influence of Anæsthetics . . . . . 57
Chloroform . . . . . 57
Ether . . . . . 58
Morphia . . . . . 59
Injection of Curari . . . . . 59
Injection of Amyl Nitrite . . . . . 59
Discussion of Previous Results . . . . . 60
Injection of Acids and Alkalies . . . . . 62
Injection of Brain Extracts . . . . . 63
Epileptic Fits . . . . . 64
Injection of Absinthe . . . . . 64
Injection of Strychnine . . . . . 64
Injection of Supra-renal Extract . . . . . 66
Effect of Ice applied to the Head and Abdomen . . . 67
Principles of the Cerebral Circulation . . . . 67
The Cerebral Blood Volume almost an Invariable Quantity . 67
A Rise of Arterial Pressure increases the Cerebral Blood Flow 68
Fallacy of the Contrary Doctrine . . . . . 68
Schema of Cerebral Circulation . . . . . 69
Intracranial Tension . . . . . 71
Past Methods of Investigation . . . . . 71
Intracranial and Cerebral Venous Pressures . . . . 73
Range of Variation . . . . . 73
Mechanism of Regulation of the Cerebral Circulation . 74
Cerebral Congestion . . . . . 75
Summary of Section III. . . . . 76

SECTION IV.—INFLUENCE OF THE FORCE OF GRAVITY ON THE CIRCULATION OF THE BLOOD.

PAGE

History . . . . . 78
METHOD OF RESEARCH . . . . . 81
Estimation of Intracranial Tension in Man . . . . 81
HYDROSTATIC AND DYNAMIC MOMENTS . . . . . 83
NORMAL EFFECT on the Carotid Artery . . . . . 84
on the Respiration . . . . . 85
on the Femoral Artery . . . . . 85
Effect on the Intracranial Tension . . . . . 86
Compensation of the Hydrostatic Moment by the Dynamic Moment . . . . . 87
In the Monkey . . . . . 87
EFFECT ON THE VENOUS CIRCULATION . . . . . 88
on the Cerebral Venous Circulation . . . . . 90
INFLUENCE OF ANÆSTHETICS . . . . . 93
Chloroform . . . . . 94
Ether . . . . . 95
EFFECT OF VASO-MOTOR PARALYSIS . . . . . 98
Section of Dorsal Cord . . . . . 98
of Splanchnic Nerves . . . . . 98
Effect of Curari and Amyl Nitrite . . . . . 98
Effect on the Respiration . . . . . 98
Effect of Bandaging the Abdomen . . . . . 99
Excitation of Divided Spinal Cord . . . . . 102
of Splanchnic Nerves . . . . . 102
INFLUENCE OF ASPHYXIA . . . . . 102
of Bandaging the Abdomen during Asphyxia . . 102
INFLUENCE OF INJECTING SUPRA-RENAL EXTRACT . . . 102
DISCUSSION OF EXPERIMENTAL RESULTS . . . . . 102
SYNCOPE . . . . . 103
The Arteriometer . . . . . 105
Asthenia . . . . . 105
Compensation for Gravity, the Clinical Key to the Vaso-motor Mechanism . . . . . 106
SHOCK . . . . . 106
Methods of Rapidly abolishing the Compensation for Gravity . 107
Cheyne Stokes and Traube-Hering Curves . . . . 108

PAGE
Influence of Position after Severe Hæmorrhage . . 108
Injection of Normal Saline after Hæmorrhage . . . . 108
Danger of Administering Chloroform in the Vertical Position . . . . . 108
Refutation of the Conclusions of the Hyderabad Commission . 109
Causes of Death from Chloroform . . . . . 110
Methods of Relieving Chloroform Collapse . . . . 110
Influence of Opening the Skull on the Cerebral Circulation . . . . . 111
Influence of Position in Heart and Lung Disease . . 111
Abdominal Belts . . . . . 111
Loss of Memory in the Vertical Position . . . . 112
Rabelaisian Effects of Fear . . . . . 112
Summary of Section IV. . . . . 113

## Section V.—CEREBRAL ANÆMIA.

History . . . . . 117
Methods of Investigation . . . . . 117
Ligature of the Four Cerebral Arteries . . . . 122
in Rabbits . . . . . 122
in Cats . . . . . 123
in Dogs . . . . . 123
in Monkeys . . . . . 124
Symptoms of Acute Cerebral Anæmia . . . . . 125
Causes of Death . . . . . 125
Pressor Effect of Closing the Four Cerebral Arteries . . 130
Depressor Effect of Re-opening One Carotid . . . . 130
Unstable Condition of Bulbar Centres after Ligature of the Four Cerebral Arteries . . . . . 130
Cheyne Stokes and Traube-Hering Curves . . . . 130
Dependence of Respiration on Blood Pressure . . . . 132
Cheyne Stokes Respiration in Hybernating Animals . . 133
Production of Cerebral Anæmia by Forcible Injection into One Carotid Artery . . . . . 135
Remote Effect of Ligature of the Four Cerebral Arteries in the Dog . . . . . 137
Symptoms of Idiocy . . . . . 137
Diary of the Animal's Condition . . . . . 137

PAGE
Recovery of the Animal . . . . . 139
Post-mortem Injection of the Circulation after Ligature of the Four Cerebral Arteries . . . . . 139
Pathways of Anastomosis . . . . . 139
Effect on the Retinal Circulation . . . . . 140
Remote Effects in the Monkey . . . . . 141
Cerebral Anæmia in Man . . . . . 141
Compression of Common Carotids in Man . . . 142
Cortical Spasm Produced thereby . . . . . 142
Ligature of the Carotid Arteries in Man . . . . 143
Epilepsy . . . . . 144
Refutation of the Cerebral Vaso-motor Spasm Theory . 145
Bulbar Spasms . . . . . 145
Cortical Spasms . . . . . 145
Theory of Epilepsy . . . . . 146
Sleep . . . . . 146
Cerebral Anæmia and Venous Congestion in Sleep . . 147
Vaso-motor Centre, the Hub round which Turns the Wheel of Active Mental Life . . . . . 148
Theory of Sleep . . . . . 148
Summary of Section V. . . . . 149

## Section VI.—METABOLISM OF THE BRAIN.

Production of Heat in the Brain . . . . . 152
Fallacies of Previous Investigations . . . . . 152
Determination of the Exchange of Blood-Gases in Brain and Muscle . . . . . 154
The Brain not a Seat of Active Combustion . . . . 155

## Section VII.—CEREBRAL COMPRESSION.

History . . . . . 156
Symptoms of Apoplexy . . . . . 160
Discussion of Past Researches . . . . . 162
Method of Investigation . . . . . 164
Compression by Continuous Injection of Fluid at a Constant Pressure . . . . . 165
Discussion of Results . . . . . 168
Escape of Injection Fluid . . . . . 168

PAGE

Translocation of the Brain and Blocking of the Isthmus Tentorii Cerebelli and Foramen Magnum . . . 169
General and Local Compression . . . . . 175
Production of Hæmorrhage in the Cranial Cavity . . 175
Non-absorption of Blood-clot . . . . . 175
Effect on Intracranial Tension and Cerebral Venous Pressure . 178
Symptoms of Apoplexy, produced by Cerebral Anæmia . . 180
Effect on Bulbar Centres of Local Hæmorrhage . . . 181
Compression by a Foreign Body of Limited Volume . . 181
Pressure Discontinuity between the Chambers of the Cranio-vertebral Cavity . . . . . 182
Pressure not Equally Transmitted by Brain in all Directions . 182
Major Symptons of Apoplexy produced by Compression of Bulbar Centres . . . . . 182
Symptoms of Compression . . . . . 184
Epileptic Fits . . . . . 184
Inhibition of Heart . . . . . 185
Spasm of Respiration . . . . . 185
Pressor Effect . . . . . 186
Causes of Death from Compression . . . . . 187
Traube-Hering and Cheyne Stokes Curves . . . . 187
Remote Effects of Intracranial Hæmorrhage . . . 188
Congestive Œdema . . . . . 189
The Circulus Vitiosus . . . . . 189
Inflammatory Œdema . . . . . 189
Circulus Vitiosus established by a Rise in Arterial Tension . 191
Depletion as a Form of Treatment . . . . . 193
Tumours of Brain . . . . . 194
Mechanism by which Compression is Produced . . . 196
Tumour and Cerebral Œdema . . . . . 197
Meningitis and Cerebral Abscess . . . . . 197
Meningocele . . . . . 198
Serous Apoplexy, Fallacy of . . . . . 199
Summary of Section VII. . . . . 199

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Bibliography . . . . . 201
Index of Name . . . . . 207

# LIST OF ILLUSTRATIONS.


| FIG. | | PAGE |
|---|---|---|
| 1. | A. Brain-Pressure Gauge | 9 |
| | B. Instrument for producing Local Compression of the Brain | 9 |
| 2. | Section of Dog's Cranium showing Torcular Herophili | 11 |
| 3. | Key and Retzius' Injection of Subarachnoid Space | 25 |
| 4. | Methods of Investigating Cerebral Circulation | 41 |
| 5. | Excitation of Central End of Upper Dorsal Spinal Cord | 44 |
| 6. | Effect of Drawing a Ligature round the Neck | 47 |
| 7. | Excitation of Peripheral End of Vagus | 48 |
| 8. | Excitation of Peripheral End of Vagus; Cerebral Venous Exits partly blocked | 52 |
| 9. | Effect of Asphyxia | 56 |
| 10. | Influence of Amyl Nitrite | 60 |
| 11. | Epileptic Fit produced by Absinthe | 65 |
| 12. | Schema of Cerebral Circulation | 69 |
| 13. | Instrument for Investigating Intracranial Tension in Man | 82 |
| 14. | Effect of Gravity, Carotid Artery | 84 |
| 15. | Effect of Gravity, Femoral Artery | 85 |
| 16. | Effect of Gravity, Carotid Artery and Intracranial Tension | 86 |
| 17. | Compensation for Hydrostatic Effect of Gravity in Monkey | 87 |
| 18. | Effect of Gravity on the Cerebral Venous Circulation | 90 |
| 19. | Influence of Chloroform on Gravity Effect | 92 |
| 20. | Influence of Chloroform on Gravity Effect | 94 |
| 21. | Influence of Ether on Gravity Effect | 96 |
| 22. | Effect of Gravity after Section of Upper Dorsal Spinal Cord | 98 |
| 23. | Influence of Bandaging Abdomen | 99 |
| 24. | Influence of Bandaging Abdomen | 100 |
| 25. | Ligature of Four Cerebral Arteries in Rabbit | 122 |
| 26. | Ligature of Four Cerebral Arteries in Cat, Cheyne Stokes Respiration | 126 |

FIG. PAGE
27. Ligature of Four Cerebral Arteries in Cat, Primary Vaso-motor Paralysis . . . . . 127
28. Ligature of Four Cerebral Arteries in Cat, Primary Respiratory Failure . . . . . 128
29. Pressor Effect of Closing Four Cerebral Arteries—Depressor Effect of Opening One Artery . . . . . 129
30. Instability of Bulbar Centres after Closure of Four Cerebral Arteries . . . . . 131
31. Cheyne Stokes Respiration and a Plethysmographic Record of Arm in Man . . . . . 134
32. Optic Disc of Dog with Four Cerebral Arteries Ligatured . 140
33. Methods of Producing Cerebral Compression . . . 156
34. Compression by Injection of Fluid into Cranial Cavity . 168
35. Section of Cranium . . . . . 170
36. Section of Cranium . . . . . 171
37. Section of Cranium . . . . . 172
38. Compression by Hæmorrhage into Cranial Cavity . . 178
39. Effect of Compression on Cerebral Venous Pressure . . 179
40. Local Compression—Vagus Inhibition . . . . 185
41. Local Compression—Pressor Effect . . . . . 186

# THE CEREBRAL CIRCULATION.

## INTRODUCTION.

The study of the exact attributes of the cerebral circulation, and how and why, from interference with the physical conditions of the normal cranio-vertebral contents, pathological states may arise, is one of the most difficult and at the same time one of the most interesting questions with which a physiologist can grapple. The hydrodynamical conditions of the cranium are so complex, and the methods of research surrounded, as they are, with many difficulties, are so exceedingly liable to lead to error, that a man's wits must indeed be about him that he may walk straight through so tangled a pathway. The whole difficulty of the subject depends upon that shell of rigid bone, a shell inexpansile, unyielding to atmospheric pressure, enclosing a brain of which the nervous substance is utterly incompressible. From the outer world of the body into the base of the cranial cavity pass pulsating arteries. These arteries permeate that membranous capsule of the brain, the pia mater, dip into the nervous matter and end in

an all-pervading capillary meshwork. Between the various arteries coursing over the surface of the pia mater there is free communication; by injection into one tributary the entire arterial system can be here readily filled. The injection at first passes rapidly through the main stems, and it is only when these have become distended that the meshwork in the pia begins to fill. From this capillary meshwork, fine branches run into the brain substance, and thence arise veins, which, trending towards the roof of the cranium, there pass into the large venous sinuses, emissaries of which once more gain the outer world. The brain and the blood which circulates through it fill the cranial cavity almost entirely. In the space that remains is the cerebro-spinal fluid, a fluid of peculiar constitution which moistens the surface of the brain. In the vertebral canal, where it bathes the spinal cord, it is relatively more in amount. The nervous substance of the brain is incompressible as water: Grashey has experimentally determined this point: the living brain full of blood is as expressible as a sponge.

The research on which I have been continuously engaged for the last four years was suggested to me by Professor Burdon Sanderson, and the fruits of this research, such as they are, which I shall endeavour to lay before you in these lectures, owe their origin primarily to him who planted the seed, whether in fruitful or unfruitful soil time and trial will tell.

It is so easy by incomplete and faulty experiment and hasty generalisation to throw discord into the

accepted pathology of the clinician, that the experimental method has often and most deservedly been met by him with incredulity and passed over with neglect. That the experimental method is a true standard by which clinical pathology can be measured goes without saying. It is the complete carrying out of the method that alone fails. Such a subject as the hydrodynamics of the cranium can only be investigated by experiment. By experiment the theory of the clinician and the conclusions of the morbid anatomist can be tested and the hand of the surgeon guided. It is as fatal to depend on results gained from the dead animal or from the use of artificial schemata, as to discuss the physics of this question without appeal to experiment at all. In the hands of many workers this method has led to fallacy, since they failed to realise that the physical conditions of the cranial cavity and of the circulation are not wholly known, and therefore no scheme or model can adequately represent them. Experiment on the living animal solely can throw light on these complex questions, and schemata are of value in so far only as they illustrate the facts gained by experiment.

The method of working I have followed has been experimental. I have worked out each step in research and have then, and only then, appealed to the evidence of the literature of the subject. In this way I have found that many of my results had been forestalled by former workers. Although such a method may lead to some personal disappointment, yet I believe it to be of the greatest value, because every one of the con-

clusions which I have finally reached is corroborated by my own independent and unbiassed experiment. If I have failed anywhere in acknowledging the work of others, it is from ignorance and not by intention.

## SECTION I.

### THE PULSATIONS OF THE BRAIN.

Observations on the movements of the brain are to be found recorded in the earliest medical writings. Galen and his followers thought that by the diastole of the brain, the πνεῦμα, the spirit of life was sucked in through the cribriform plate and mingled with the vital spirit which ascended by the arteries from the heart. For, in the words of a Galenist, "the heart, the fountain of life, doth boil up out of the finest parcel of blood, a little flame called a vital spirit, and it panteth by reason of its heat incessantly." In the laboratory of the cerebral ventricles the animal spirits were prepared from this mixture and driven by the systole of the brain into the nerves. "There the animal spirit doth work local motion whilst running up and down through the nerves and filling the muscles; it on this side stretcheth tendon, on that side it pulls them back. But being weary it seeks rest, and therefore, having left the organs of sense, hideth itself into its retirements, which thing we call sleep; it refresheth itself in the brain, and running up and down its cells, upon what vain visions it light reassumes them to be viewed over which we call a dream." The waste pro-

ducts from the preparation of these animal spirits were, it was thought, excreted by the nose and the infundibulum. Apoplexy was attributed to the stoppage of this excretion. Such was the doctrine held by some of the Galenists.

It was supposed by others, that the dura pulsated, and, by driving onward the animal spirits, produced movements of the soul and of the muscles. Thus Baglivius and Pacchionus thought that the dura mater was a muscular and contractile membrane, whilst Willis and Mayow discovered, what they held to be, the contractile fibres of this membrane. In ascribing the movements to the pulsation of the arteries in the dura, Fallopius came nearer to the truth ; Ridley, a clear-headed anatomist of the seventeenth century, ascribed the pulsation entirely to the cerebral arteries. De la Mure, Haller and Lorry, by finally establishing the cardiac and respiratory origin of the cerebral pulsation, proved that the dura possessed no independent power of movement in itself, and showed that the collapse of the chest-wall pressed upon the thoracic veins, and drove back the venous blood, thus causing the expiratory expansion of the brain.

In recent times Donders, denying that any movements of the brain could take place in a closed skull, opened up a fresh phase of discussion and inquiry. He trephined an animal and observed a pulsation in the vessels of the pia. He then fixed into the hole a glass window, and found that all sign of pulsation ceased. Experimenting on the same lines, Leyden also denied to the brain any movement in the closed skull. Longet

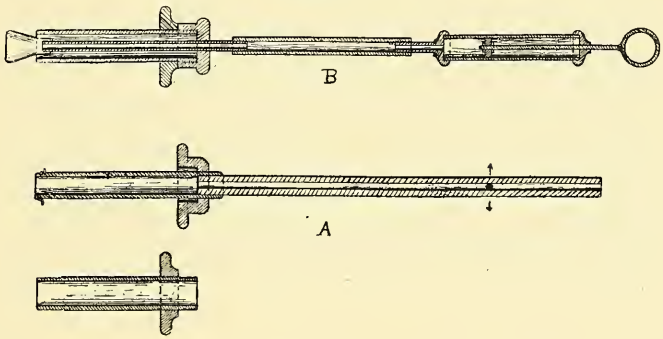
went still further, and denied that even the cerebral arteries could pulsate. The reason given for this strange assertion was that the brain completely filled the rigid unyielding box of the skull in which it lay enclosed. Schultz, however, by employing a better light to illuminate the cranial window, was able to discern and bear witness to the pulsations of the vessels in the closed skull. In the last twenty years the cardiac and respiratory movements of the brain have been observed and recorded by Leyden, Jolly, Salathé, Franck, Oudin, Mosso and many others. Records have been taken from animals, from men with cranial defects, from infants by means of the fontanel. With a tambour, Bergmann recorded the movements in the brain of a woman whose skull was defective. On compressing both carotids the pulsation ceased. The cardiac expansion of the brain was synchronous with the systole of the heart, the respiratory expansion with expiration. Salathé recorded simultaneously the cerebral movements, the respiration, and the blood pressure in the carotid. Franck observed a great expansion of the brain on forced expiration, and noted that the cerebral movements were comparable to those perceived in a limb enclosed in a plethysmograph. Duret was the first experimenter to record the pulsations of the cerebro-spinal fluid within the closed cranio-vertebral cavity. This he did by means of a tambour placed in apposition to the occipito-atlantal ligament. Donders and Berlin thought that when the cranial cavity was closed in proportion to the expansion of cerebral arteries the cerebral veins collapsed,

and on each diastole of the arteries cerebro-spinal fluid was absorbed, to be once more transuded on systole. In these two ways space was made for the cardiac systolic increase of the arteries, and the brain mass did not as a whole either expand or contract. Bergmann pointed out that in the cardiac systolic phase the capillary pressure is at its highest, and therefore transudation rather than absorption would occur. Althan showed that the errors of Donders and Berlin arose from neglect of the fact that the brain pulsates in a direction where resistance is least. Thus when the cranium is trephined and the dura opened, the pulse can scarcely be seen in the occipito-atlantal membrane, for the trephine hole has now become the seat of least resistance. Similarly, when a trephine hole is closed by a glass window, the occipito-atlantal membrane becomes the seat of least resistance, and a pulsation appears there. Franck very neatly exhibited this phenomenon by recording the pulsations of a limb placed in a plethysmograph. He fixed two tubes into the chamber of the instrument, and stretched across one of these tubes a diaphragm of thin india-rubber. The pulsations of the limb could now be seen in the open tube only, but if this were closed the elastic membrane in the other tube immediately responded to the movement.

I will now turn to my own method of demonstrating the movement of the brain in the closed cranium. I trephine a skull in the parietal region, worm the trephine hole with a mechanic's tap, carefully divide the dura without causing any hæmorrhage, and screw

a piece of steel tubing into the hole. Over the end of a piece of brass tubing of smaller diameter than the steel tube, but of equal length, I tie a piece of the thinnest india-rubber membrane (Fig. 1, A). This second tube is passed into the steel tube, and the two are screwed together by means of a male and female screw. By means of this mechanism the india-rubber membrane comes into exact apposition with the brain. Attached to the end of the brass tube is a piece of glass tubing of a fine bore. This is connected with a

FIG. 1.




T-piece, one branch of which leads to a pressure bottle, and another to a mercury manometer. The whole apparatus is completely filled with water, and a bubble of air, to act as an index, is introduced within the fine-bore glass tubing. Before the apparatus is screwed into the steel tube the normal no-pressure position of the air index, when the tube is held horizontally and the membrane is flat, is marked on the glass tube. On screwing in the apparatus the air index, which is at first forced out, is again, by raising the pressure bottle, brought to the normal position. That is to say, the

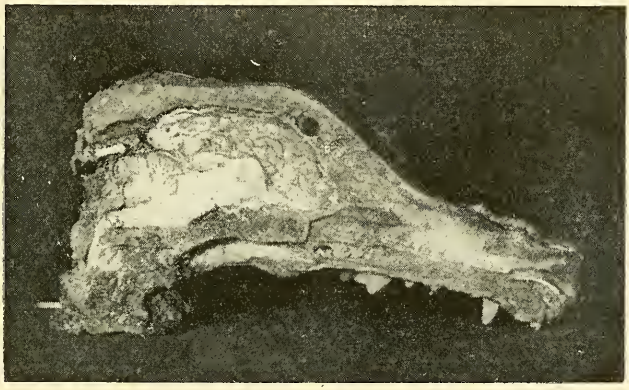
brain, which is slightly bulged outwards by the intracranial pressure, is forced into that flattened position which it naturally must assume against the closed cranial wall. The elevation of the manometer by this method gives me the normal intracranial tension. The pressure of the cranial contents against the skull wall is exactly balanced, and the skull becomes once more a closed cavity. The air index in the cerebral pressure gauge—as I shall call this instrument—perfectly exhibits cardiac and respiratory undulations of intracranial pressure. Now very frequently on opening the dura there is scarcely a drop of fluid to be seen between this membrane and the pia mater. The brain, therefore, is in close apposition to the dura, and we may rest assured that the brain itself pulsates in the closed cranium. This diastole and systole of the brain are made possible in the unyielding box of the cranium in two ways.

(1) By the ebb and flow of the cerebro-spinal fluid. The occipito-atlantal and other vertebral ligaments extend in cerebral diastole and allow the fluid to escape from the cranial cavity, while in systole, through the elasticity of these ligaments coming into play, it is driven back. To what extent this can take place I shall show in the next section.

(2) By compression of the cerebral veins in diastole another mechanism is provided. Cramer was the earliest recorder of the cerebral venous pressure. He placed a cannula in a branch passing from the transverse sinus into the jugular vein, and noted a transmission of the pulse into this vein. Gaertner and

Wagner reached the same result by observations of the blood-flow from the same vein. This method of obtaining cerebral venous pressure is exceedingly inconvenient, and entails large dissections in large animals, and even in such failure is frequent owing to clotting. The method which I have used to investigate the cerebral venous circulation is as follows. In

FIG. 2.



a dog the superior longitudinal sinus with its tributaries opens into a large venous cavity within the occipital protuberance. This bony cavity is the torcular Herophili. The transverse sinuses lie within the osseous part of the tentorium cerebelli and lead out of this cavity. Part of the blood finds its exit by the post-glenoid foramen and reaches the external jugular vein, but a large portion passes into the large sinuses which run down the vertebral canal. It is quite simple to drill a hole into the torcular, and then immediately to screw into the hole a brass tube filled with magnesium sulphate solution. This brass tube is

connected with a water manometer. In Fig. 2 is shown a photograph taken from a longitudinal section of a dog's head immediately after death. A hole drilled into the torcular is marked out white in the figure. The manometer can be directly observed, or connected with a delicate tambour to record the movements. In this way a continuous record of cerebral venous pressure can be obtained with little dissection and considerable ease in dogs of small size. The manometer exhibits all the cardiac and respiratory pulsations, the latter in a very marked way. The brain is an organ which pulsates with every stroke of the heart and with every respiratory movement, and owing to its peculiar conditions, enclosed as it is in an unyielding box, the cardiac pulse is not entirely spent in distending the arterioles and capillaries, but is transmitted to the venous sinuses. The brain, as is shown by the cerebral pressure gauge, is lifted up by the stroke of the arteries at its base, and is thrown against the cerebral veins. By the rise of pressure in the right side of the heart, the blood in expiration is dammed back into the sinuses, and the brain must then expand more at the expense of the cerebro-spinal fluid. We find the contrary to occur in inspiration, the veins are emptied by the suction action of the thorax, the brain collapses, and the cerebro-spinal fluid re-enters the cranial cavity.

The brain resembles a limb or a kidney placed in a plethysmograph or an oncometer. Expansion of the organ, due to the arterial stroke, is transmitted to the cerebro-spinal fluid and to the blood in the venous

sinuses. These fluids take the place of the oil in the oncometer. The brain, unlike the kidney, expands more during expiration than during inspiration. This is due to the absence of efficient valves in the cranial and vertebral veins, and the immediate continuity of these veins with the right auricle and with the venæ cavæ. Symington has shown that the supposed valvular action of the oblique openings of the cerebral veins into the longitudinal sinus does not exist. Fluid can easily be injected from the sinus into these veins. I have found the same thing. Symington also doubts the competency of the valves of the jugular vein in man. I shall show, in a later section, that in animals the cerebral venous pressure rises millimetre for millimetre with the rise in the thoracic veins. Normally then the brain is affected by expiratory rise of general venous pressure; this is in contrast with such an organ as the kidney which follows the inspiratory rise of general arterial pressure. If the inspiratory arterial rise becomes in any way increased so as to be greater than the venous expiratory expansion, then the respiratory curve of the brain is reversed (Figs. 18 and 37). The movements of the brain can become pathologically increased or diminished. Any cause that produces increased tension of the dura may, when the skull is trephined, stop that membrane showing pulsation. I have found that whenever the arterial blood pressure and intracranial tension rise the movements are increased in the brain pressure gauge. The cerebral spinal fluid then vacates the skull and the gauge or cerebral venous manometer exhibits the

increased pulsation produced by the heightened arterial tension. If there be within the cranium a foreign body of a certain volume, then capillaries are obliterated and intracranial tension becomes, as I shall show later, raised and the movements in the gauge are correspondingly increased. If, on the other hand, the blood pressure be very low and the cranium open the pulsation may be increased, because the brain may partly empty during the expiratory fall of arterial pressure and collapse from the trephine hole, to swell out once more during the inspiratory rise. The brain pulsation is least marked in conditions of normal pressure.

The comparison which I have just now drawn between the cranio-vertebral cavity and an oncometer has been previously recognised by Naunyn, Falkenheim, and Dean. The method adopted by these authors to investigate the cerebral circulation was to pass into the subarachnoid space a catheter which they connected with a manometer, and thus the vertebral canal became for them the chamber of an oncometer. The success of such a method depends entirely on two factors : first, on the amount of fluid within the oncometer ; and, secondly, on its absolute freedom from leakage. Imagine a kidney in an oncometer to expand to such a degree that all the oil be driven out of the chamber. Necessarily any further efforts at expansion on the part of the organ cannot be recorded by the instrument, for the kidney everywhere comes in contact with the rigid walls of the oncometer. I have found this in the case of the brain. The cerebro-spinal fluid within the cranium is insufficient in

amount, and the brain on expanding comes everywhere into contact with the cranial wall. Falkenheim and Naunyn's method cannot therefore be made use of in studying the cerebro-circulation. I have also found that fluid leaks from the cranio-vertebral cavity. Therefore on this account also the oncometric method may prove fallacious. This leads me to the discussion of the next section of my work—namely, the cerebro spinal fluid. I would first, however, summarise the conclusions I have already reached.

## SUMMARY OF THE CEREBRAL PULSATION SECTION.

(1) The movements of the brain are proved to exist in the closed cranium.

(2) These movements are of cardiac and respiratory origin.

(3) The brain stands in such close relationship to general venous pressure that, normally, its greatest expansion is in expiration and not in inspiration. The cardiac pulse is transmitted to the cerebral veins.

(4) Owing to the ebb of cerebro-spinal fluid into the less rigid vertebral canal, as well as to the diminution of the calibre of the cerebral veins and sinuses, the brain is able to expand to a limited extent.

(5) Any increased tension of the dura mater decreases its exhibition of the cerebral pulse.

(6) The cerebral pulsation is least when the intra-cranial tension is normal.

## SECTION II.

### CEREBRO-SPINAL FLUID.

MAGENDIE first laid stress upon the study of the cerebro-spinal fluid and discovered the foramen named after him which puts the intra-ventricular cavities in connection with the subarachnoid space. Up to recent times it was considered that the subdural space in the cranium and the subarachnoid space in the vertebral canal were continuous. It was thought that the arachnoid supplied a parietal layer to the dura and a visceral layer to the pia mater. Key and Retzius by their use of coloured gelatine for injecting at very low pressures, have conclusively shown that in a man the subarachnoid space of the vertebral canal is not continuous with the subdural space of the cranial cavity. On the other hand, there is a continuity of the subarachnoid space traceable from the vertebral canal to the cisternæ at the base of the brain and thence along the course of the cerebral vessels up to the Pacchionian bodies of the longitudinal and other sinuses. This is the true cranial subarachnoid space. These authors in their fine monograph demonstrated that the Pacchionian bodies formed a channel of communication between the subdural, the subarachnoid

spaces, and the venous sinuses, and that these peculiar bodies were not only found in the longitudinal but in the cavernous sinus at the base of the brain. Each Pacchionian villus is covered with a membrane continuous with the arachnoid. Outside is another fine membranous sheath derived from the dura mater, and the interval between the two is continuous with the subdural space. Fluid injected by these authors under low pressure into either the subdural or the subarachnoid space found its way into the sinuses through those bodies. Previous to the work of Key and Retzius the transmission of powdered cinnabar from the subdural space to the sinuses through the Pacchionian bodies had been demonstrated by Quincke. Key and Retzius evidenced the continuity of the subarachnoid space with the lymph spaces in the optic, auditory, and other nerves. Michel and Schwalbe succeeded in injecting the lymphatics of the neck from the subarachnoid space. Key and Retzius pointed out that neither the brain nor the spinal cord possessed true lymphatic vessels. The lymph finds its way out of these organs by means of perivascular spaces in the tunica adventitia of the blood-vessels ; these perivascular spaces communicate with the subarachnoid space at the surface of the brain and cord. These authors have proved : (1) That the central canal of the cord and the ventricles of the brain are connected with the subarachnoid space by the foramen of Magendie and the two other foramina in the lateral recess by the side of the flocculus cerebelli ; (2) That the subarachnoid space is continuous, extending throughout the central nervous

system; (3) That the subdural is anatomically separated from the subarachnoid space; (4) That injections into the subarachnoid or the subdural spaces pass out into the venous sinuses through the Pacchionian bodies and through the sheaths of the nerves and cranial lymphatics.

Duret, Adamkiewicz, Naunyn, Schreiber and Falkenheim have observed the rapid escape of fluid injections from the cranio-vertebral cavity. Duret recorded the escape of 583 c.c. of water in two hours. Adamkiewicz writes that "every tendency of the cerebro-spinal fluid to take up a higher tension than normal is stopped by the passing of fluid into the blood, and this resorption goes on till the tension is equal to the blood pressure." Falkenheim and Naunyn passed a catheter up the cauda equina into the subarachnoid space and allowed normal saline to run in from a pressure bottle. At a pressure of 15 mm. Hg absorption was apparent but very slow. At a pressure of 59 mm. Hg it reached 1 c.c. a minute. When a ligature was drawn round the dura at the level of the second or third cervical vertebra so as to cut off the spinal from the cranial cavity, the rate of absorption was lessened from 6 c.c. in ten minutes to ·6 c.c., the pressure of injection being 40 mm. Hg. According, then, to the result of this experiment the cranium is the chief seat of absorption. Vieussens, Magendie, Luschka, Schwalbe, Naunyn and Schreiber have recorded observations on the spread of injection of fluid through the cranio-vertebral cavity. The first two authors were not able to inject fluid from the cerebral chamber of the cranium into the vertebral

canal, the last workers were able to do so. All of these authors agree that injections through the occipito-atlantal ligament can spread upwards into the cranial cavity and cerebral ventricles and downwards into the vertebral canal.

I have myself made many experiments on the absorption of fluids from the cranio-vertebral cavity. On trephining the lamina of the vertebral column and screwing into it a tube, I have found normal saline can be driven into the vertebral canal. If a second trephine hole be made in the parietal region none of the injection into the vertebral canal can be driven out from this hole. The mid-brain is floated up by the fluid into the isthmus of the tentorium cerebelli and plugs this up, while the great brain likewise moves upwards and plugs up the trephine hole in the parietal region. If, on the other hand, the saline be injected into the parietal hole it can in most cases be driven through the cranio-vertebral canal and out of the hole in the vertebral column. The whole central nervous system can thus be freely irrigated. In some cases, and nearly always if the pressure of the injecting fluid be high and applied suddenly, this cannot be done. By the pressure of the fluid the cerebrum is driven into the isthmus of the tentorium cerebelli and blocks this up so that no fluid can be expelled. If the cerebrum be removed from the cranium of a dog and the empty cerebral chamber be filled with water, none of the fluid leaks through the foramen magnum. This shows how completely the cerebellar chamber is filled by the brain mass within it. I have found that saline injected

at any pressure above the cerebral venous pressure disappears from the cranio-vertebral cavity ; the higher the pressure the more rapid its disappearance. As a result of injecting saline coloured with methyl blue, fluid can be traced passing straight into the venous sinuses. In so short a time as ten to twenty minutes the blue colour may be found secreted in the stomach and in the bladder. On the other hand, the lymphatics in the neck in so short a time are not even coloured. After an hour's steady injection, the deep cervical and lymphatic glands are seen to be only partly tinged with the blue colour. Serum passes through into the veins with as much ease as normal saline. It is clear that the rapid absorption of fluid from the cranio-vertebral cavity takes place by means of the veins and not by way of the lymphatics. On examination the power of the dura mater to prevent filtration through its structure has not proved to be great. Over the sinuses at the base of the brain of a dog or cat the dura is stretched tight and thin as the parchment over a drum. If a fresh dura be carefully stripped from the cranial wall and with the serous surface innermost, be tied over a tube filled with saline, and this tube be in connection with the pressure bottle and a manometer, it will be found that beads of fluid will slowly exude through the membrane at a pressure of 10 to 20 mm. Hg. Through a human dura mater filtration took place at a pressure of 40 mm. Hg. In the same way by moderate pressures fluid can be driven through the pericardium and the parietal layer of the peritoneum, while the central tendon of the diaphragm withstands a

pressure of 100 mm. Hg. One little fact which came out in this research on filtration through membranes, is that of the extraordinary extensibility and elasticity of the parietal layer of the peritoneum. In this respect, the peritoneum stands in sharp contrast to the dura mater or pericardium. In the physiology of the circulation and respiration, this fact is perhaps of some little importance. From these results it is clear that fluids can slowly pass from the cranio-vertebral cavity directly into the veins at a pressure just above the cerebral venous pressure and with increased facility at higher pressures. At moderate pressures filtration can take place directly through the dura into the venous sinuses. I will give the particulars of a typical experiment proving some of the above conclusions.

EXPERIMENT.

*Dog.*—Anæsthetic, morphia.

Cerebral venous pressure in torcular Herophili = 108 mm. $H_2O$.

Pressure of cerebro-spinal fluid under occipito-atlantal membrane = 108 mm. $H_2O$.

Saline injected at the occipito-atlantal membrane or through a trephine hole in the parietal region was absorbed from the cranio-vertebral canal at a pressure just above 108 mm. $H_2O$.

Immediately after death, water injected into the parietal hole under considerable pressure reached the vertebral canal but very slowly, and ran out from a hole in the lumbar region.

Water injected at the lumbar opening floated up the

brain against the parietal hole and did not escape thence.

Water injected into the cranio-vertebral cavity could be seen to escape from the torcular Herophili.

On injecting water into the parietal hole under considerable pressure after first cutting away the occipito-atlantal ligament, the brain could be seen descending into the foramen magnum, so that the tip of the calamus scriptorius came to lie below the foramen magnum. The water leaked round the brain but very slowly, owing to the descent of the great brain into the isthmus of the tentorium cerebelli, and of the cerebellum and medulla against the margin of the foramen magnum.

The excised dura mater allowed water to filter through it at so low a pressure as 70 mm. $H_2O$.

The ease with which fluid can pass at low pressures from the parietal region to the spinal canal depends on the pulsation of the brain, for the cerebral diastole allows the fluid to find a pathway along the course of the shrinking vessels. This is shown by the lines of intense staining after the injection of methyl blue. When the injection pressure is high the brain is driven into the isthmus tentorii cerebelli, and the cerebral circulatory tension so heightened that the diastole is rendered ineffectual. The pathway of filtration of fluid from the cranial cavity may be through the Pacchionian bodies, through the dura, or through the veins in the pia mater. By increased intracranial pressure, even supposing the pial veins to be collapsed,

yet at the point of their entry into the rigid sinuses, an incollapsible portion must exist where filtration can readily take place.

Under normal pressures the secretion and absorption of cerebro-spinal fluid does no doubt follow osmotic laws. Under abnormal pressures both secretion and absorption become filtration. Thus, Halliburton and others have found that cerebro-spinal fluid when originally drawn off from the subarachnoid space has a peculiar chemical composition, but on being drawn off again and again after the refilling of the space it equals more nearly the composition of serum. The same change has followed in the repeated tappings of the sacs of spina bifids and hydrocephalics.

Subsequent to the publication of my work on the pathway of absorption, Reiner and Schnitzler have independently brought confirmatory evidence. They collected the blood from the jugular vein and injected potassium ferrocyanide in saline solution into the cranium. This salt very rapidly appeared in the jugular vein. The venous flow was quickened by the injection, not slowed. On the other hand, injection of olive-oil caused compression of the cerebral vessels and slowed the venous outflow. However, on microscopical examination some oil could be seen in the venous blood. As this experiment succeeded in rabbits some other pathway than the Pacchionian granulations must be substituted, for these bodies do not exist in these animals.

We may conclude then that fluid will not remain for any length of time in the cranio-vertebral cavity at a

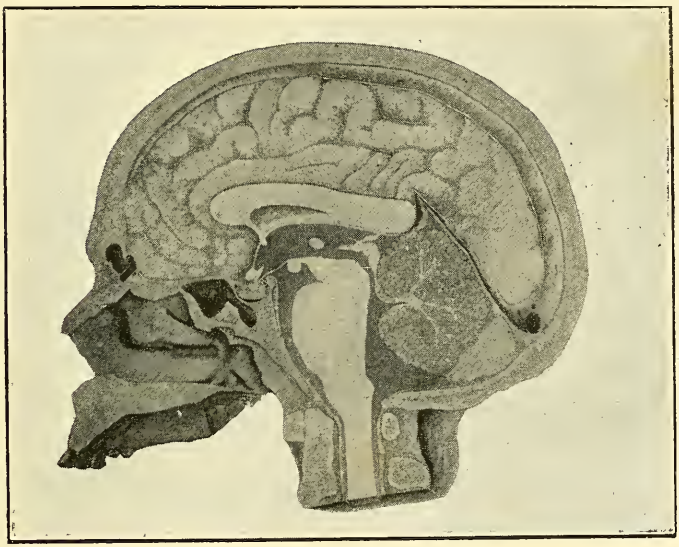
pressure above the cerebral venous pressure. How important a fact this is in the pathology of cerebral pressure I will attempt to show later. From my experimental results the lack of continuity between the subarachnoidal and the subdural space cannot be held to be physiologically important. By gradual injections at low pressures and with the greatest care *only* could Key and Retzius show the continuity of the true cerebral with the spinal arachnoidal space.

On opening the dura in the living animal with the utmost care the fluid within the cranium, if any, is in the subdural space. It is impossible to so open the dura as to see the subarachnoid space. Saline or serum injected subdurally runs with ease into the subarachnoid space of the vertebral column at a pressure just above intracranial pressure.

The plates in Key and Retzius' monograph which are copied into most anatomical works of to-day give, I believe, an entirely erroneous idea of the contents of the cranium in the living animal. In Fig. 3, taken from one of these plates, we find the arachnoid cisternæ at the base of the brain distended with an artificial injection, and the tentorium pushed upwards and out of place. In the living animal the chief contents beside the brain substance is blood, and not cerebro-spinal fluid. I have never been able in the scores of living animals I have examined to find, on opening the skull in various places, more than a very slight amount of cerebro-spinal fluid in the cavity, and no such injection of cisternæ and ventricles as Key and Retzius picture. The living brain with its circulating blood almost

entirely fills the cranium, and the fluid that moistens its surfaces is little more in amount than the synovial fluid in a joint. The cerebral subarachnoid space I believe to be chiefly a potential rather than an actual space, except in those few places where inequalities of the brain surface are rounded off by small collections of fluid beneath this membrane.

Fig. 3.



The subdural space, although undoubtedly anatomically separate from the subarachnoid, as I have seen both in man and animals, is yet, I believe, physiologically one with it. Either by foramina or by filtration, fluid passes with the greatest ease from one space into the other. The whole evidence of pathology is in favour of this view. Such a statement as this from the most recent text-book of anatomy is entirely misleading: "Coloured fluids injected into the subdural space are

never found to pass into the subarachnoid space, the arachnoid limiting membrane being everywhere a closed one."

On the question of the secretion of the cerebro-spinal fluid I have less information. In the normal healthy state the amount of fluid is very slight. Every physiologist knows how impossible it is to collect any quantity of it from the dog, for instance.

On opening the occipito-atlantal membrane in the animal, and on placing its head downwards, two to five c.c. at the most will be obtained. The largest collection of this fluid is around the medulla oblongata and beneath the occipito-atlantal membrane. On trephining over the cerebrum in animals often not a drop of fluid is to be seen between the dura and the brain, in other cases only a slight moisture. Bochefontain has drawn attention to this fact.

Cotugno made a hole in the lumbar region of the spinal column, lifted up the head of the corpse and collected the outflowing liquid. He found that in twenty different experiments on the human body about five ounces of cerebro-spinal fluid could be obtained. This amount is no doubt far above that which exists in the living, normal man. The fluid is increased by post-mortem changes.

Haller injected water into the arteries of a dead animal with the result that the water exuded from the surface of the brain. On injecting coloured gelatine into the veins this fluid appeared in the ventricles. He states also that some hours after death fluid collects in the ventricles, and if wiped away will reappear.

Bergmann injected defibrinated blood at a high pressure (800 to 1200 mm. Hg) into the peripheral end of the carotid of a horse, and found that the flow of lymph from the cervical lymphatics was somewhat, although not conclusively, increased during the time of injection.

Falkenheim and Naunyn carried out some experiments on the rate of secretion of the cerebro-spinal fluid. On continuously tapping off the fluid from dogs they found 36 to 240 c.c. secreted in the twenty-four hours. The rate of secretion seemed uninfluenced on raising the arterial pressure by compression of the abdominal aorta. Injection of saline into the circulatory system, however, raised the secretion by 50 per cent.

In turning to the study of cerebral circulation, I shall show that the cerebral capillaries stand in much closer relationship to venous than to arterial pressure. From observations I have myself made, I believe the rate of transudation of the fluid depends directly on the cerebral venous pressure. The cerebro-spinal fluid is normally maintained at the tension of the cerebral veins. If the fluid be allowed to escape from the cranio-vertebral cavity, the pressure within this cavity then falls to zero, while the cerebral venous pressure remains unaltered. In consequence transudation from the capillaries becomes continuous, and hence the large amount of fluid obtained in cases of fracture of the skull. Its rate depends upon the capillary blood pressure. High arterial tension increasing transudation, coupled with high venous tension preventing

absorption, is the most favourable condition for making greater the outflow of cerebro-spinal fluid. Such a condition is experimentally found in the spasms of strychnine. The pressure of the cerebro-spinal fluid can never mount above that of the cerebral veins on account of the rapid absorption that takes place through these veins. From this section I may now summarise the following conclusions.

### SUMMARY OF THE CEREBRO-SPINAL FLUID SECTION.

(1) That the subdural and subarachnoid spaces, although anatomically separate, are physiologically one.

(2) That fluid escapes directly into the veins from the subdural and subarachnoid space at any pressure above the cerebral venous pressure. The tension of the cerebro-spinal fluid and the cerebral venous pressure are normally the same.

(3) That the brain and spinal cord can be irrigated with fluid at a low pressure. This can be effected through one trephine hole without any counter-opening, and is a perfectly harmless operation.

(4) That the brain with its circulating blood almost entirely fills the cranial cavity in the living animal.

(5) That the cerebro-spinal fluid preserves its peculiar constitution in normal conditions of pressure only. If it be drawn off, its place is taken by a serous transudation.

(6) That the rate of this transudation directly depends on the difference between cerebral venous pressure and the subarachnoid pressure. Diminished

subarachnoid pressure produces increased transudation until the cerebral venous pressure and the cerebro-spinal fluid pressure are again equalised.

(7) That no pathological increase of cerebral tension can be transmitted by the cerebro-spinal fluid, because this fluid can never be retained in the meningeal spaces at a tension higher than that of the cerebral veins.

(8) Finally, it is suggested that in such a pathological condition as meningitis irrigation of the meninges might be employed. The operation could be as easily and safely carried out as that of irrigation of the peritoneum.

## SECTION III.

### THE CEREBRAL CIRCULATION.

#### MONRO-KELLIE DOCTRINE.

The methods hitherto employed in researches on cerebral circulation are, in one way or another, incomplete and defective. Important questions remain undecided. Such problems as these suggest themselves. Does the volume of the blood within the brain vary in amount, or does it remain constant, "cabin'd, cribb'd, confin'd," by the wall of the skull? Is the brain matter exposed to the full tension of the varying blood pressure, or is there existent some compensatory mechanism by means of which the intracranial pressure is kept constant? Is the brain supplied with vaso-motor nerves which regulate its blood supply, or does it, in the varying conditions of life, passively endure every to-and-fro swing of the general blood pressure? Lastly, does a rise of arterial pressure increase the supply of blood to the brain, or does it, as some have supposed, cause an anæmia? For it is conceivable that in the closed box of the cranium cerebral capillaries may be obliterated by the expansion of the large arteries, since the tension of the

vessels at the base of the brain may be directly transmitted through the brain substance.

In 1783 Alexander Monro the younger put forward the view that the quantity of blood within the cranium is almost invariable.

"For being enclosed in a case of bone," he writes, "the blood must be continually flowing out of the veins that room may be given to the blood which is entering by the arteries. For, as the substance of the brain, like that of the other solids of our body, is nearly incompressible, the quantity of blood within the head must be the same, or nearly the same, at all times, whether in health or disease, in life, or after death, those cases only excepted in which water or other matter is effused or secreted from the blood-vessels; for in these a quantity of blood equal in bulk to the effused matter will be pressed out of the cranium."

Monro's teaching was supported by Abercrombie and Kellie, and became known as the Monro-Kellie doctrine.

"The cranium," Abercrombie writes, "is a complete sphere of bone, which is exactly filled by its contents, the brain, and by which the brain is closely shut up from atmospheric pressure and all influences from without, except what is communicated through the blood-vessels which enter it. Upon the principles of hydraulics it seems probable that the vessels of the brain must always contain a considerable quantity of blood, even when other parts of the system are exhausted of it. In such a cavity as the cranium the blood probably cannot be diminished below a certain quantity, unless something entered it to supply its

place, and, in the language of the old philosopher, 'prevent a vacuum'."

Kellie put the doctrine to the test of experiment. He killed many animals by bleeding, opened the skull, and found the brain still to contain blood. If he trephined the animals before bleeding, the brain, under the influence of atmospheric pressure, became empty of blood. He arrived at the conclusion that the blood contents of the brain in the closed cranium could only vary in so far as serous fluid increased at the expense of the red blood. Burrows repeated Kellie's experiments, and came to an opposite conclusion. He insisted on the greater importance as a mechanism of the cerebro-spinal fluid, which allowed the volume of the blood in the brain to vary. Burrows, however, admitted the constancy of the cranial contents. He writes: "We know that in health the quantity of serous fluid that exists in the ventricles, membranes and substances of the brain is considerable. Regarding this serum as an important element of the contents of the cranium, I admit that the whole contents of the cranium—that is, the brain, the blood and this serum together—must be at all times nearly a constant quantity."

It was held by Reid that Burrows magnified the influence of cerebro-spinal fluid. "It is well known," he writes, "that there is found very little of the cerebro-spinal fluid—in fact, in general little more than what is sufficient to moisten the surface of the membranes—in the interior of the cranium in healthy persons up to the middle of life. Under these circumstances, the

quantity of cerebro-spinal fluid that could be displaced in the interior of the cranium must be trifling."

Many observers have repeated these experiments of Kellie and Burrows with opposite results. Engel, among others, declares that hanging human corpses by the head or the heels makes no difference to the cerebral blood content.

Kussmaul and Tenner came to the conclusion that on this point no certain information can be drawn from post-mortem examination, and with this I fully agree. For, "if an individual die during a partial convulsion the brain may be found blanched; while if death supervene some time after the convulsive attack is over, a state of extreme hyperæmia is just as likely to be met with."

The examination after death of the condition of the vessels is a very deceptive method of obtaining information as to the amount of blood in the brain prior to death.

If a corpse be placed in a position with the head higher than the trunk, and the cranium opened, the brain under the influence of gravity and atmospheric pressure collapses and empties itself of blood. If the head be placed lower than the trunk a contrary condition results. The whole blood content of the brain may change therefore at the moment when the pathologist opens the skull.

Again, if the large veins of the thorax and head and neck be engorged with blood as is the case after death from asphyxia, or from hypostatic congestion, then on opening the skull the brain may be found filled with blood from the pressure in these veins. Another way

by which the blood content can change after death under the influence of gravity, is by the passage of gas from the blood into the intracranial cavity, in the same way as gas collects in the arterial system after death.

Again, after death the capillary walls become damaged, and serous fluid as well as gas may filter through in the direction of least pressure under the tension due to the elasticity of the blood-vessels which strive to return to the no-tension position. Thus the vessels collapse and serous fluid increases. The same thing may happen before death, when the head is elevated and the blood pressure very low, for then, as I shall point out in dealing with the influence of gravity on the circulation, the cerebral circulation practically ceases before the general circulation. Thus, in some men who died of cold in a snowstorm, Kellie found more than four ounces of serous fluid in the ventricles of the base of the brain. In these various ways the volume of the blood in the brain and of gas and serous fluid may be altered by post-mortem changes. Hence post-mortem examinations in reference to the truth of the Monro-Kellie doctrine appear absolutely contradictory in result.

I am certain that no sure evidence of the cerebral blood volume can be gained by post-mortem examination. Anatomists should remember in drawing conclusions as to the comparative size of brains, that the weight of this organ may vary largely on account of those post-mortem changes which I have described.

I will now turn to the experimental proof of the Monro-Kellie doctrine in the living animal.

Donders observed the capillaries of the pia mater in the live animal through a glass window which he fixed into a trephine hole, and found that with the skull thus completely closed, the amount of injection of the capillaries varied. This, however, is no proof of the variation in the total volume of blood within the cranium. It may only indicate a variation in the relative distribution of the same quantity of blood. Thus, if the capillaries are expanded the veins may be proportionately compressed. Donders' method gives no proof in either direction. Kussmaul and Tenner agree with the observation of Donders, but admit that the venous sinuses could not be observed. They conclude that the cerebral blood volume can vary, while, on the other hand, their experiments give me corroboration of my own experimental conclusion, which is, that in the living animal the Monro-Kellie doctrine is true. On compressing the innominate and left subclavian arteries, these authors observed through the glass window in the cranium that the arterioles disappeared, and the small veins became less. The brain, however, did not collapse or retreat from the window. If, on the other hand, the glass window was faultily placed and allowed leakage into the cranial cavity, the air passed within and the brain collapsed under atmospheric pressure. This experiment proves that the brain in the closed cranium can by no means completely empty itself of blood even though the blood pressure should fall to zero. The collapse of the smaller pial vessels may be caused either by increase of the amount of cerebrospinal fluid at the base of the brain, or by increase of

blood in the venous sinuses, or through both of these causes together. I have reached this conclusion by another method. If the spinal cord be divided, or the splanchnic nerves be cut and the animal be placed in the vertical, feet down position, the blood pressure in the brain will, under the influence of gravity, fall to zero. If the skull be now trephined and the dura be rapidly opened, the brain which was before in close apposition with the dura may now be seen collapsing under one's very eyes, as it is emptied of blood by atmospheric pressure. We may rest assured, therefore, that in all physiological conditions the blood content of the brain can vary only to a slight degree by the ebb and flow of cerebro-spinal fluid, and that the Monro-Kellie doctrine is to all intents and purposes true. Further evidence on this point will be manifest in dealing with the circulation of the brain. In pathological states of cerebral compression, however, as I shall show later, large alterations occur in the volume of blood in the brain.

I will now proceed to the methods of investigating the cerebral circulation.

v. Schultén's list of the possible means include:

(1) The direct observation of the pia mater.

(2) A record of the volume of the brain.

(3) A measurement of intracranial pressure.

(4) Measurement of the blood pressure in the cerebral vessels.

(5) A measurement of the velocity of blood flow in the cerebral vessels.

All these methods have been tried by different observers.

Nothnagel, Schüller, and Ackermann simply inspected the vessels of the pia mater under varying conditions of experiment. This is a most untrustworthy method, for the brain is exposed and the cranium is not closed as in the normal condition, and the vessels in the exposed portion of the brain rapidly suffer from inflammatory congestion, and therefore changes in calibre may take place in them which cannot take place in the closed cranium.

v. Schultén and Salathé registered changes in intracranial pressure by screwing a tube into the skull, and this tube they connected with a manometer. Now I have shown that with any increase of intracranial pressure the brain floats up against the trephine hole and acts towards it as a valve, so that no more fluid can escape into the manometer. Thus it is impossible, by injecting fluid into the subarachnoid space of the vertebral canal, to drive it out of a hole in the cranium.

Another method is to use the cranio-vertebral cavity as an oncometer. This was first employed by Falkenheim and Naunyn, and later, in a modified form, by Dean. These investigators connected the spinal subarachnoid space with a manometer by means of a catheter. Falkenheim and Naunyn passed this catheter up the lower end of the dural sac in the region of the cauda equina, and by Dean it was passed up the sheath of a spinal nerve.

Now I have already shown that when the intracranial pressure rises the small quantity of cerebro-

spinal fluid within the skull cavity passes into the spinal canal. The base of the brain then descends and blocks up the isthmus tentorii cerebelli and the foramen magnum, and no further rise of cerebral pressure can be communicated to the manometer which is in connection with the spinal subdural space.

Knoll's method meets with the same objection. He measured the pressure in the spinal subarachnoid sac by passing a cannula through the occipito-atlantal membrane, and this he connected with the manometer.

By no one of these methods can the changes in intracranial pressure be completely measured. The *sign* of varying pressure can at most be shown, not the amount. The brain, on its increased expansion, closes up, not only a trephine hole in the roof of the cranium, but the foramen magnum in the base. The intracranial pressure may therefore vary exceedingly, but the manometers of not one of these investigators can fully record the variations.

The results of the last four authors cannot even be regarded as true records of variations of pressure in the contents of the vertebral canal, for fluid leaks from the subarachnoid space.

Of considerable value is Gaertner and Wagner's method of recording the outflow of blood from the lateral sinuses, but their results, although of great interest, are not conclusive, because they did not simultaneously record the arterial and venous pressures with the cerebral venous outflow.

Hürthle, and more recently Cavazzani, have recorded the blood pressure in the circle of Willis. This they

did by placing a cannula in the peripheral end of the carotid artery after tying all but the cerebral branch, and at the same time recording the pressure in the central end of the carotid artery.

Cybulski, by means of his instrument the photo-hæmatochometer, investigated the rate of flow in the internal carotid in the varying conditions of experiment.

None of the investigators whom I have mentioned have made records in their researches of the general venous pressure as well as the arterial pressure. Roy and Sherrington, by an ingenious plethysmographic method, recorded the variations of the vertical diameter of the brain with the cranium open. At the same time they registered the arterial pressure, and in some cases they investigated the general venous pressure in similar conditions of experiment. Their method seems incomplete in so far as that the normal condition is not maintained, since throughout the experiments the cranium is open. In the closed cranium large alterations of blood volume are impossible. In the open cranium, with the atmospheric pressure brought to bear upon the cerebral capillaries, the physical conditions of the cerebral circulation are altered. These authors, moreover, do not seem to have recorded simultaneously with the general venous pressure the variations of the blood volume and the arterial pressure. In their search for cerebral vaso-motor nerves they did not do the most essential experiment; that is, after division of the spinal cord in the dorsal region, the stimulation of either the vaso-motor centre or the central end of the spinal cord. Nevertheless, their work

has yielded valuable results on many points. Wertheimer has recently investigated the cerebral circulation by a similar method to that of Roy and Sherrington.

My object has been to make the method of research complete by simultaneously recording the arterial pressure, the general venous pressure, the intracranial pressure and the cerebral venous pressure, the cranium being as in the normal condition a closed cavity. I shall show that the record of general venous pressure is absolutely essential, and all previous researches in which this has been neglected cannot be held conclusive on any single point. In a large portion of this research on the cerebral circulation I have had the advantage of my friend, Mr. Bayliss, as a co-worker. Since the publication of our joint paper I have developed some new experiments, and re-confirmed many of the old ones.

## METHOD OF RESEARCH.

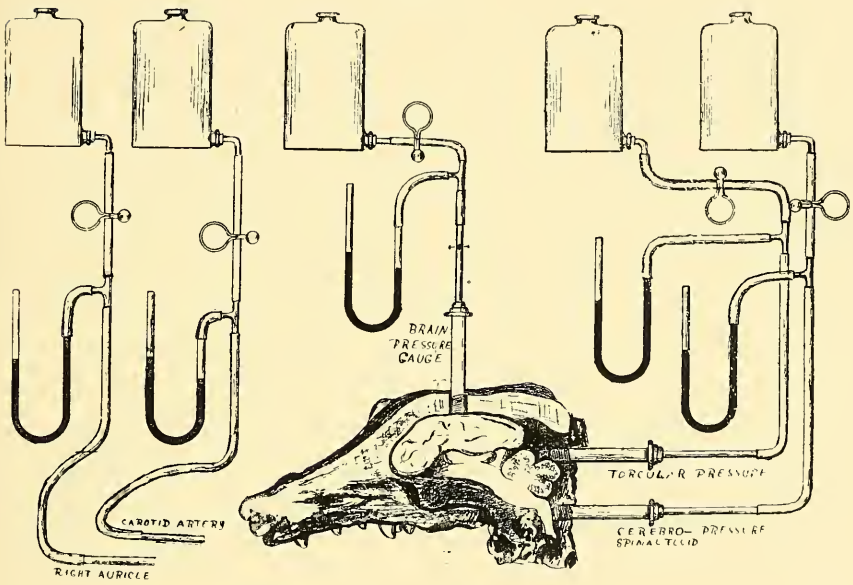
The animals employed for this research were in most cases dogs. Morphia was the anæsthetic generally used, but the results have been controlled by the use of other anæsthetics. In no single case has morphia been found to obscure results, while ether and chloroform frequently do so.

The method of procedure of the experiments was as follows (Fig. 4):

A cannula was placed in the central end of the carotid artery. A second long cannula was passed down the external jugular vein, and on the same side into the right auricle. The torcular Herophili was

trephined and a third cannula was screwed into the hole thus made. The cannula in the carotid artery was placed in connection with a mercurial manometer, and the other cannulæ with manometers filled with a saturated solution of magnesium sulphate. These venous manometers were observed directly on a milli-

FIG. 4.




metre scale, or were connected with delicate tambours or piston recorders. Thus, records were obtained of the general arterial pressure, of the general venous pressure, and of the cerebral venous pressure. In many experiments the intracranial pressure—that is, the pressure in the subdural space—was also recorded. This was necessary to prove the fact, which always holds true, that the intracranial pressure or cerebral tension and the cerebral venous pressure are, in all

physiological conditions of the circulation, the same. In these investigations it is absolutely necessary to eliminate any passive effects caused by changes in the general venous or arterial circulation. Such is the method of record used by Bayliss and myself to investigate the existence of cerebral vaso-motor nerves. We carried on this research in the following ways:

### SEARCH FOR CEREBRAL VASO-MOTOR NERVES.

(1) We divided the vago-sympathetic nerves and stimulated the central end in both the curarised and the uncurarised animal. Curari was employed to remove the passive effect of the respiratory spasms produced by stimulation of this nerve. In such an investigation it is absolutely necessary to eliminate any passive effects caused by changes in the general venous or arterial circulation.

(2) We divided the spinal cord in the upper dorsal region at a level of the second to the sixth dorsal nerves, so as to abolish the passive effects produced by constriction in the splanchnic area, and we stimulated either the central end of the cord or the vaso-motor centre in the bulb.

(3) We planned a new method of stimulating the cervical sympathetic nerve, so as to include any of its possible branches to the vertebral arteries together with the supply to the carotid arteries. In the experiments carrying out this last method the animals were anæsthetised and then lightly curarised, and artificial respiration supplied. The thorax was then opened in the middle line, and weighted hooks were used to keep

the walls of the cavity well apart. The heart was protected from exposure by a pad of cotton-wool. The sympathetic nerve was then caught up—with an aneurism needle—where it lies high in the thorax, and as it passes over the first rib just below the stellate ganglion, and a ligature was passed under it and tied. The nerve was then divided below the seat of the ligature, and a pair of long shielded electrodes were placed on the nerve between the ligature and the stellate ganglion. Finally, the ligature was tied on to the electrodes in such a way that the nerve could not be displaced, and the thorax was then closed. By this means was ensured the stimulation of the whole of the sympathetic fibres which supply one side of the head and neck. The accelerating fibres to the heart were also included.

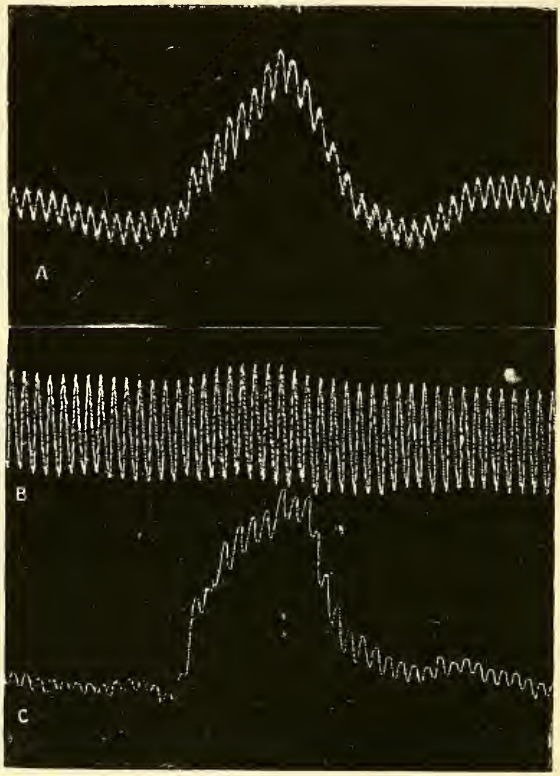
By all these methods of research we found that the cerebral venous pressure (and the intracranial pressure) passively followed the slightest changes in the general arterial and general venous pressure. We could obtain no evidence whatever of any independent change pointing to the existence of any vaso-motor nerves supplying the brain. I will now give typical examples of the experimental results.

(1) Excitation of the central end of the vago-sympathetic trunk produces either a rise or a fall in cerebral venous pressure, according as there is in the ascendency the sensory constrictor influence or the depressor action of the nerve on the vaso-motor centre.

(2) After division of the spinal cord and upon stimulating the central end of the divided cord, or the vaso-

motor centre, there is a slight rise of general arterial pressure, and the cerebral venous pressure passively follows this rise. The general venous pressure may remain unaltered or may suffer an insignificant rise or fall.

FIG. 5.




A: Carotid. B. Right Auricle. C. Torcular.

In Fig. 5 is shown the effect of stimulating the central end of the cord after it has been divided at the level of the third or fourth dorsal vertebra. On dividing the cord still higher at the level of the second dorsal vertebra the effect, although very much slighter, was still the same in character.

(3) On stimulating the stellate ganglion in the thorax, acceleration of the heart and a slight rise of arterial pressure occurs; the general venous pressure may fall slightly or remain unaffected while the cerebral venous pressure follows the arterial rise. (For this tracing see *Journal of Physiology*, 1895.)

Thus, Bayliss and I have been entirely unable to find any evidence of a vaso-motor supply to the brain. Each of these experiments has been many times repeated, and the injury to vaso-motor excitability by shock or the use of curari has been carefully prevented by slight curarisation, by keeping the animals on warm baths and by rapid experimentation. We have stimulated the whole of a sympathetic supply and the whole of the cranial nerve supply, and we have by dividing the spinal cord diminished the overpowering passive effect of the general circulation to a vanishing point. Yet we have obtained no positive results. The cerebral venous pressure has never varied in contrary sense to the general circulatory pressures. In a recent and exhaustive research, Gulland has failed to demonstrate by every known histological means the existence of any vaso-motor nerves in the pial vessels.

Turning to the evidences of past workers, I find that Nothnagel and Ackermann obtained at times, on stimulating the cervical sympathetic, evidence of dilatation of the pial vessels.

Recently Cavazzani found evidence of both cerebral constrictor and dilator fibres in this nerve. The methods of these workers are valueless in deciding such a question. Schultz, Riegel and Jolly, Cramer,

v. Schultén, Gaertner and Wagner, Hürthle, Roy and Sherrington have obtained no positive evidence of any active cerebral effect from stimulation of this nerve. There is, therefore, overwhelming evidence that there are no cerebral vaso-motor nerves in the cervical sympathetic. In the hands of Jolly, Stilling, and Dogiel no active effect on cerebral circulation has been produced in rabbits by stimulation of the depressor nerve.

We have further investigated the result on the circulation of the brain of a number of other effects.

### EFFECT OF DRAWING A LIGATURE ROUND THE NECK.

If a cannula be placed in the trachea and a ligature be then drawn round the neck so as to exert gentle pressure on the veins, there is an immediate marked and sustained rise of cerebral venous pressure (Fig. 6).

Directly the ligature is withdrawn the pressure again falls to normal.

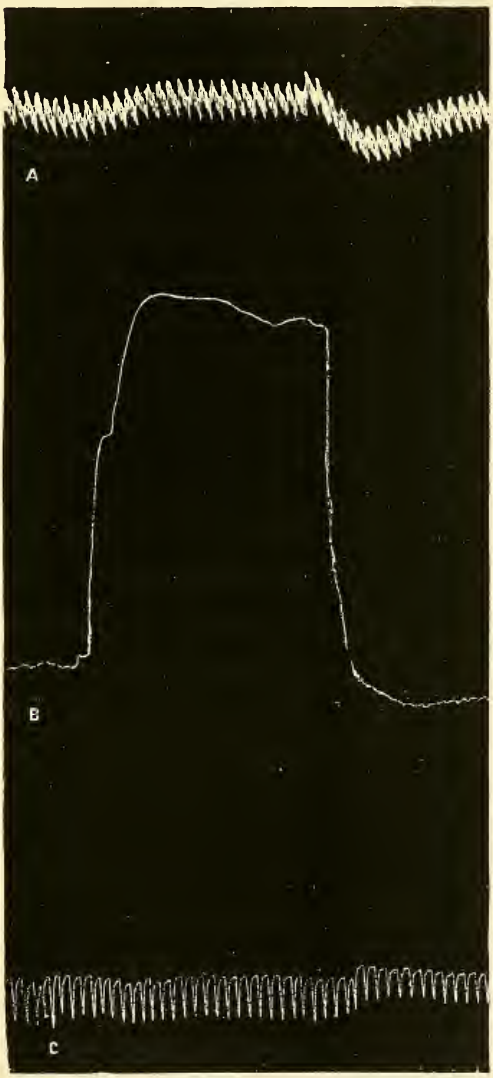
In our general experimental method we occluded one carotid artery and one jugular vein. This makes little difference to the cerebral circulation. Occlusion of both carotids causes a fall in cerebral venous pressure. To this point I shall return in discussing cerebral anæmia.

### STIMULATION OF THE PERIPHERAL END OF THE VAGUS AND DISCUSSION OF THE PHYSICAL CONDITIONS OF THE CEREBRAL CIRCULATION.

These experiments have led to very interesting results which help to explain the physical conditions of the cerebral circulation. If one vagus nerve be divided

and the peripheral end stimulated, the arterial pressure rapidly falls, while the general venous pressure as

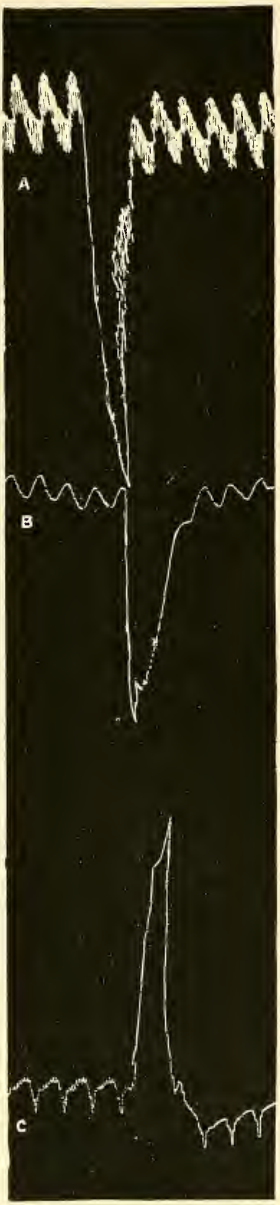
FIG. 6.




A. Carotid. B. Torcular. C. Right Auricle.

rapidly rises to the mean pressure of the whole circulatory system (Fig. 7).

FIG. 7.




A. Carotid. B. Torcular.
C. Right Auricle.

If the animal be horizontal and the hydrostatic effect of gravity at its least, the intracranial pressure or cerebral venous pressure will also be the same as the mean pressure. In determining capillary pressure, the venous pressure is of equally great importance as the arterial pressure. Following in the steps of the school of Ludwig, Bayliss and Starling in this country have lately insisted on the fact that the capillary pressure stands in closer relationship to the venous than to the arterial pressure. In general, so long as the systemic venous pressure remains constant, but only so long, the intracranial pressure—and that is the cerebral capillary pressure—follows the arterial pressure. If both alter, the intracranial pressure is affected by both, and an alteration of pressure of equal amount and in opposite directions in both will affect the intracranial pressure from the venous side to a far greater degree than from the arterial

side, because it is on the arterial side that the resistance lies. The cranio-vertebral veins possess no efficient valves. Symington has shown that the cerebral veins can be injected from the longitudinal sinus, and the obliquity of the openings of these veins does not, as has been suggested, act as a valvular arrangement. Symington has also expressed suspicion of the competency of the valves in the jugular veins of man. I have myself determined that if the torcular Herophili be opened in the freshly killed animal and the abdominal and thoracic veins be compressed, venous blood can be driven out of the torcular in a continuous stream. If in the living animal the torcular venous pressure and the general venous pressure be simultaneously recorded in manometers placed side by side, and if at the same time the abdomen be compressed, then the pressure rises in both by equal increments—that is to say, when the arterial pressure remains constant and by greater increments in the torcular if the arterial pressure rises also. Thus the pressure in the cranio-vertebral veins varies absolutely as the pressure in the venæ cavæ. The intracranial pressure also varies absolutely as the pressure in these veins. On the other hand, the torcular and intracranial pressures do not vary absolutely as the general arterial pressure, but only in the same direction, because these pressures are equal to the arterial pressure minus the unknown resistance which opposes the tension of the vascular walls on the arterial side. Thus the variations of arterial pressure read in millimetres of mercury only produce variations in cerebral venous pressure which can be read in millimetres of water.

The exact method of experiment by which I have arrived at these important results is simultaneous record of—

(1) Arterial pressure in the central carotid.

(2) General venous pressure in the right auricle.

(3) Cerebral venous pressure in the torcular Herophili.

(4) Cerebro-spinal fluid pressure taken by trephining the atlas and screwing a tube into the hole and connecting this with a manometer; or, intracranial pressure taken by means of the cerebral pressure gauge.

The general arterial and venous pressures are then made to vary by compression of the abdomen, by vagus inhibition of the heart, and by drugs, such as amyl nitrite and suprarenal extract.

On compressing the abdomen the intracranial and general venous pressures rise absolutely together. On the other hand, employing suprarenal extract after section of the vagi, I have obtained in a typical experiment a rise of—

126 mm. Hg in arterial pressure;

31 mm. Hg in intracranial pressure;

10 mm. Hg in cerebro-spinal fluid pressure.

The last figure proves that the cerebro-spinal fluid within the cranium is not sufficient in amount to allow complete expansion of the brain, and that the brain becomes actually pressed against the cranial wall and its tension not fully recorded by that of the cerebro-spinal fluid. The cerebro-spinal fluid leaks from the cranio-vertebral cavity.

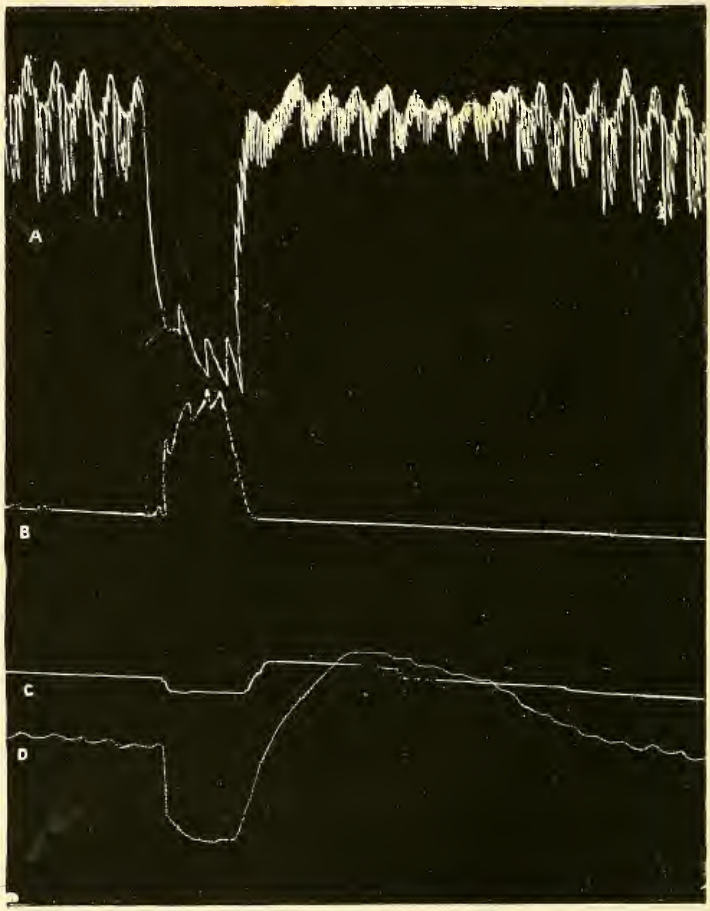
Some of Cramer's experiments support mine. He

found on ligaturing the veins of the head and neck that cerebral venous pressure rose in the proportion of 1·5 to 1 or even 2·5 to 1. On ligaturing the abdominal aorta the carotid pressure rose in the proportion 2 to 1, the cerebral venous pressure 1·5 to 1. These results show, therefore, that a rise in general venous pressure is more effectual in influencing cerebral venous pressure than a vastly greater rise in arterial pressure.

There is another point in the mechanism of the cerebral circulation. The cerebral vessels enclosed in the unyielding box of the cranium cannot expand to any great degree. When the limit of expansion is reached they become of the nature of rigid tubes, inexpansile. Now any alteration of pressure in a circulatory scheme produced by a pump reaches its maximum more rapidly in a rigid tube than in an elastic tube. If great resistance lies in the outlet of a rigid tube, on varying the force of the pump pressure will rise still much more rapidly and fall much more slowly there. When the outlet is closed entirely, then the pressure will rapidly rise to the static pressure of that part of the schema in which the rigid tube is placed. Such an effect is shown on many of our tracings in cases where the cerebral venous outlet has been accidentally impeded either by clotting or kinking of the veins arising from the position of the head; or where it has been experimentally produced by gently tightening a ligature round the neck or by plugging the lateral sinuses. Thus in the next tracing cerebral venous pressure and intracranial pressure are seen on stimulating the

peripheral vagus to fall somewhat when the heart ceases to beat, and to rise much above the normal when the heart begins again to beat, and finally to fall

FIG. 8.




A. Carotid. B. Right Auricle. C. Intracranial Pressure. D. Torcular.

slowly back to the normal. On stimulation of the peripheral end of the vagus, therefore, we normally obtain a fall of intracranial or cerebral venous pressure if the resultant effect of the fall of arterial pressure be

greater than the effect of the rise of general venous pressure. On the other hand, if the outlet of the cerebral veins be impeded a large rise of cerebral venous or intracranial pressure always occurs when the heart again begins to beat—that is to say, the arterial pressure rapidly rises while the veins cannot rapidly empty.

Roy and Sherrington observed after vagus stimulation a marked expansion of the brain. Their tracings show exactly the same result as has been obtained by us when the cerebral venous outlet was blocked. These authors curiously ascribe this phenomenon to some constricting influence of the peripheral end of the vagus on the general venous system. They write, "that according to our observations there are in the vago-sympathetic nerves descending fibres, section or stimulation of which can produce either a rise or a fall of the general venous pressure, and these fibres can be called into action either by direct stimulation or reflexly by excitation of ascending fibres, the corresponding nerve of the other side being intact."

Dean, by his ingenious method of recording cerebro-spinal fluid pressure, never obtained any sign of this marked expansion observed by Roy and Sherrington. It is probably owing to his method that Dean never found any indication of this effect. It was unlikely that the veins of the neck would be kinked or pressed as his records were obtained from the vertebral canal, not from the brain. In Roy and Sherrington's method it is extremely probable that the venous outflow would sometimes, from experimental interference with the

head, become impeded. There is abundant evidence that these vagus effects are entirely mechanical in origin; and for the sake of making this quite clear I will once again recapitulate.

Upon producing cardiac inhibition the rise of general venous pressure is simply caused by the pressure in the whole circulatory system reaching one dead level or mean pressure; the pump ceases to work, and the pressure in the arteries runs down and in the veins runs up until the mean is established throughout the system. There is no need to invoke the aid of any hypothetical vaso-motor nerves. In those cases in which the cerebral venous outlet is impeded, and a marked rise of intracranial pressure occurs when the heart again begins to beat, the explanation is also mechanical, and the conditions then are as follows:

(1) When the heart ceases to beat the brain cannot empty itself of blood because the cranium is a closed cavity.

(2) When the heart again begins to beat the brain is full of blood.

(3) If the venous exit be impeded the force of the heart-beat will be transmitted from the large arteries at the base of the brain to the venous sinuses, and blood will enter more rapidly than it can escape. Hence there is a great rise in the pressure of the torcular.

(4) Finally, as the blood again slowly forces open exits from the venous sinuses the pressure in the torcular once more falls to normal.

### EXCITATION OF THE SENSORY NERVE.

Stimulation of any sensory nerve causes a rise of general arterial pressure, and intracranial or cerebral venous pressure passively follows this rise. General venous pressure rises slightly or remains unaltered. Evidence of other workers support these experiments. Thus, while Nothnagel thought he observed constriction of the pial vessels, Jolly, Knoll, Cramer, Roy and Sherrington unanimously agree that the effects on cerebral circulation are passive in origin.

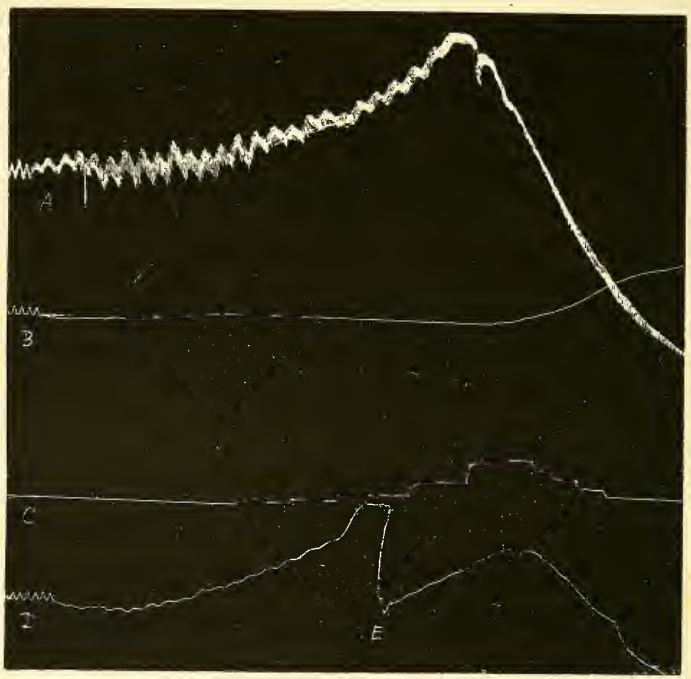
### ASPHYXIA.

On producing asphyxia in a curarised animal by stopping the artificial respiration, the general venous pressure, owing to the mechanical effect of stopping the respiratory movements, at first falls. The cerebral venous pressure passively follows this fall. During the subsequent rise in arterial pressure the general venous pressure does not vary, while the cerebral venous pressure passively follows the arterial rise. During the last stage of asphyxia when the arterial pressure is falling, the general venous pressure is rapidly rising owing to the failure of the heart. Cerebral venous pressure is now the resultant produced by the arterial and general venous pressures. Thus it is seen that when the arterial pressure has returned to the normal level the cerebral venous pressure is far above the normal, owing to the large rise that has at this time taken place in the general venous pressure.

We have never obtained the slightest evidence in

our numerous experiments in asphyxia of any change in the cerebral vessels which could not be explained passively. The historical evidence of asphyxia is of a conflicting nature, and this is naturally so seeing that

FIG. 9.




A. Carotid. B. Right Auricle. C. Intracranial Pressure. D. Torcular. At E the recording piston was lowered. Tracing reduced one-third.*

none of the experimenters have recorded simultaneously the general venous pressure with the arterial and intracranial pressures. Hürthle obtained evidence of a preliminary fall of pressure in the peripheral end of the carotid during asphyxia, which fall he

* The torcular recording instrument was more than twice as delicate as that of the right auricle.

attributed to vaso-dilatation of cerebral vessels. Falkenheim, Naunyn and Dean noticed a preliminary fall in cerebro-spinal fluid pressure, and the last attributed this to vaso-constriction of the cerebral vessels. These results are, I believe, to be attributed solely to the preliminary fall in general venous pressure, which is occasioned mechanically by the cessation of respiration. Knoll concluded that the cerebro-spinal fluid pressure rose earlier than the rise in arterial pressure, and lasted longer. v. Schultén rightly ascribed part of the rise of cerebro-spinal fluid pressure to a rise in general venous pressure; Gaertner and Wagner found no evidence of constriction of the cerebral vessels. Roy and Sherrington noticed a preliminary expansion of the brain on asphyxia, which expansion was not, they said, to be explained by any rise in general arterial or venous pressure. The general venous pressure, however, was not *at the same time* recorded in the experiments of these authors. If the animals were insufficiently curarised and moved the muscles of the neck in the slightest degree, or if in producing the asphyxia any pressure were put on the veins of the neck by clamping the trachea or detaching the artificial respiratory tube, then this preliminary expansion obtained by Roy and Sherrington might easily be so caused. Wertheimer obtained expansion of the brain on obstruction of breathing due to general venous congestion and expansion on asphyxia following a general arterial rise.

### INFLUENCE OF ANÆSTHETICS.

Administration of chloroform produces a fall of arterial pressure and a rise in general venous pressure. I have seen the general venous pressure begin to rise immediately after administration of chloroform. This may be partly due to alteration of respiration. The cerebral venous pressure temporarily follows this rise, and then the greater effect of the arterial fall. In the later stages of chloroform poisoning, as in asphyxia, there is a rapid rise in the cerebral pressure following the now greatly increased pressure in the right auricle. The general venous pressure rises because of the failure of the heart. In this case, again, as the pump ceases to act the arterial pressure falls to the mean, and the venous pressure rises to the mean. Gaertner and Wagner found an increased flow of blood from the lateral sinus during the stages of chloroform poisoning. This they ascribed to active dilatation of the cerebral vessels. I explain it, on the other hand, mechanically as due to the rise of general venous pressure consequent on the increasing cardiac and vascular paralysis. We have obtained no evidence of active dilatation of the cerebral vessels taking place during inhalation of chloroform. Roy and Sherrington thought they obtained evidence of cerebral contraction which could not be explained passively.

Ether produces a slighter fall of arterial pressure and a slight rise of pressure in the right auricle. The cerebral venous pressure follows the greater effect of the arterial fall. *In all these experiments it is important*

*to avoid any muscular movements, for these markedly affect the general and cerebral venous pressures.*

### MORPHIA.

Morphia produces no peculiar effect on the cerebral or general circulation. This is confirmed by Hürthle and Roy and Sherrington. Thus morphia is far the best anæsthetic for researches of this nature. The animals remain asleep in an absolutely constant condition, and the vaso-motor mechanism is in no way damaged by morphia as it is by ether and chloroform.

### CURARI.

By damaging the vaso-motor tone and increasing the capacity of the circulatory system the injection of curari causes a fall in all three pressures.

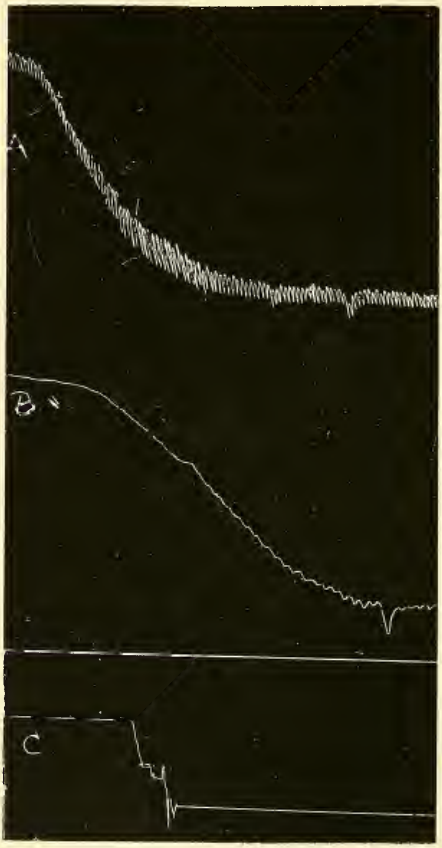
### AMYL NITRITE.

Cash and Dunstan have proved that this drug when applied only to the vaso-motor centre produces primarily vaso-constriction and rise of blood pressure. Gaskell and Shore by their cross circulation experiments reached the same conclusion in regard to chloroform. When they obtain entrance into the general circulation these drugs act on the local vaso-motor mechanism and produce vaso- and cardiac dilatation. The effect on the general and cerebral circulation of these drugs is exactly comparable. When amyl nitrite is injected into the jugular vein there follows :

(1) A rise of general venous pressure accompanied by a rise of cerebral venous pressure.

(2) A fall of arterial pressure accompanied by a fall of cerebral venous pressure. If, however, the arterial fall is not great its effect on cerebral venous pressure

Fig. 10.




A. Carotid. B. Torcular. C. Intracranial Pressure.

may by the rise in general venous pressure be almost masked. This rise of general venous pressure is doubtless due to the effect of the drug on the heart. In the tracing here given (Fig. 10) the fall of arterial tension was so great that the effect of the venous rise

was not shown. A. G. Levy has recently concluded from records of the elastic recoil of the brain that amyl nitrite caused cerebral vaso-dilatation. The method he employed was one devised by Horsley. Weights were placed on the top of a metal plunger which rested on the surface of the brain, and the descent of the plunger and the amount of its recoil on the removal of the weights were indicated by an index and dial attached to the apparatus. Levy recorded the cerebral recoil first in the normal condition, and then after the inhalation of amyl nitrite. He found that the elastic recoil of the brain was, if anything, increased by amyl nitrite while the arterial pressure fell 20 to 30 mm. Hg. On this and other experiments he has based the extraordinary assertion that the cerebral recoil does not depend on general circulatory tension, but is due primarily to the elasticity of the brain substance. This conclusion is overthrown at once by the fact that it is impossible to measure any elasticity in excised strips of brain substance. The simultaneous record of general venous pressure, which I have shown to be absolutely essential, Levy omitted, but apart from this his method is radically faulty on the following grounds :

(1) The cranium is open, and the brain therefore not in its normal physical conditions.

(2) The elasticity of the membranes is not eliminated.

(3) The plunger produces inflammation and thrombosis of the cerebral vessels, and produces œdema, as Levy himself confesses.

(4) His instrument is not proved to be sufficiently

delicate. A fall in arterial pressure of 20 to 30 mm. Hg might, according to my results, only produce a fall of at most 5 mm. Hg in cerebral capillary pressure, and Levy's instrument may not record so small a variation. Levy has himself found that variations of general venous pressure such as occur in asphyxia do markedly alter cerebral tension. I have shown that cerebral venous pressure varies millimetre per millimetre with general venous pressure, but this is by no means the case with regard to arterial variations. The enormous resistances that lie on the arterial side seem to have escaped due recognition by this author. Levy's research has value in showing how long and to what extent after expression by a foreign body the brain can recover its shape, but apart from this no other conclusions can be properly drawn as to the physical conditions of the brain.

### INJECTIONS OF ACIDS, ALKALIES AND BRAIN EXTRACTS.

Roy and Sherrington found that intravenous injection of free acids produced great and immediate expansion of the brain. Bayliss and I used lactic, hydrochloric, sulphuric and nitric acids in weak and strong solutions. We have injected these acids freely diluted, and we have injected them scarcely diluted at all. We have injected them into the central end of the jugular vein and into the peripheral end of the carotid artery. We have found that if the injection is followed by convulsive and increased respiratory movements, as is often the case, the general venous pressure from purely

mechanical causes rises, and cerebral venous pressure or intracranial pressure passively follows its rise. We have been entirely unable to confirm the results that Roy and Sherrington obtained with acids, and have not found the slightest evidence of active dilatation of the cerebral vessels. The injection of alkalies causes a fall in arterial pressure, and the cerebral venous pressure passively follows this fall. On this point we confirmed the results of Roy and Sherrington.

### BRAIN EXTRACTS.

Roy and Sherrington found that a saline extract of the brain produced a marked cerebral expansion. These authors concluded from this experiment that "the chemical products of cerebral metabolism contained in the lymph which bathes the walls of the arterioles of the brain can cause variations of the calibre of the cerebral vessels; that in this reaction the brain possesses an intrinsic mechanism by which its vascular supply can be varied locally in correspondence with local variation of functional activity." We have prepared the extract of brain according to the method of Roy and Sherrington and have injected it into many animals. We have also injected a solution of the dry grey matter of the brain prepared by Messrs. Willows, Francis and Butler. In no case have we been able to observe any active effect on cerebral circulation. If a large amount of fluid is injected or if convulsive movements are produced, or if the veins of the neck are accidentally compressed, then a marked rise of cerebral venous pressure occurs from simple

mechanical reasons. If none of these contingencies arise the effect of injecting brain extract is *nil*. We have been unable, therefore, to find evidence of any such local vaso-motor mechanism in the brain as has been indicated by Roy and Sherrington.

### EPILEPTIC FITS.

In the uncurarised animal an epileptic fit, produced by excitation of the cortex owing to its convulsive movements, produces a rise in general venous pressure. Cerebral venous pressure follows this rise. During the course of a fit the arterial pressure may both fall and rise. In the curarised animal the arterial pressure rises to a high degree, and the cerebral venous pressure follows this rise. On injecting absinthe into the curarised animal, a succession of intense clonic spasms of the arterial system ensues, general venous pressure remains practically unaltered, and intracranial or cerebral venous pressure follows exactly the arterial trace (Fig. 11).

### STRYCHNINE.

During the strychnine spasms arterial pressure rises enormously, cerebral venous or intracranial pressure follows this enormous rise, and that of the general venous pressure, which also rises largely owing to the convulsive movements. Roy, Sherrington and Wertheimer found great expansion of the brain, and Gaertner and Wagner a great increase in cerebral venous flow during strychnine spasms.

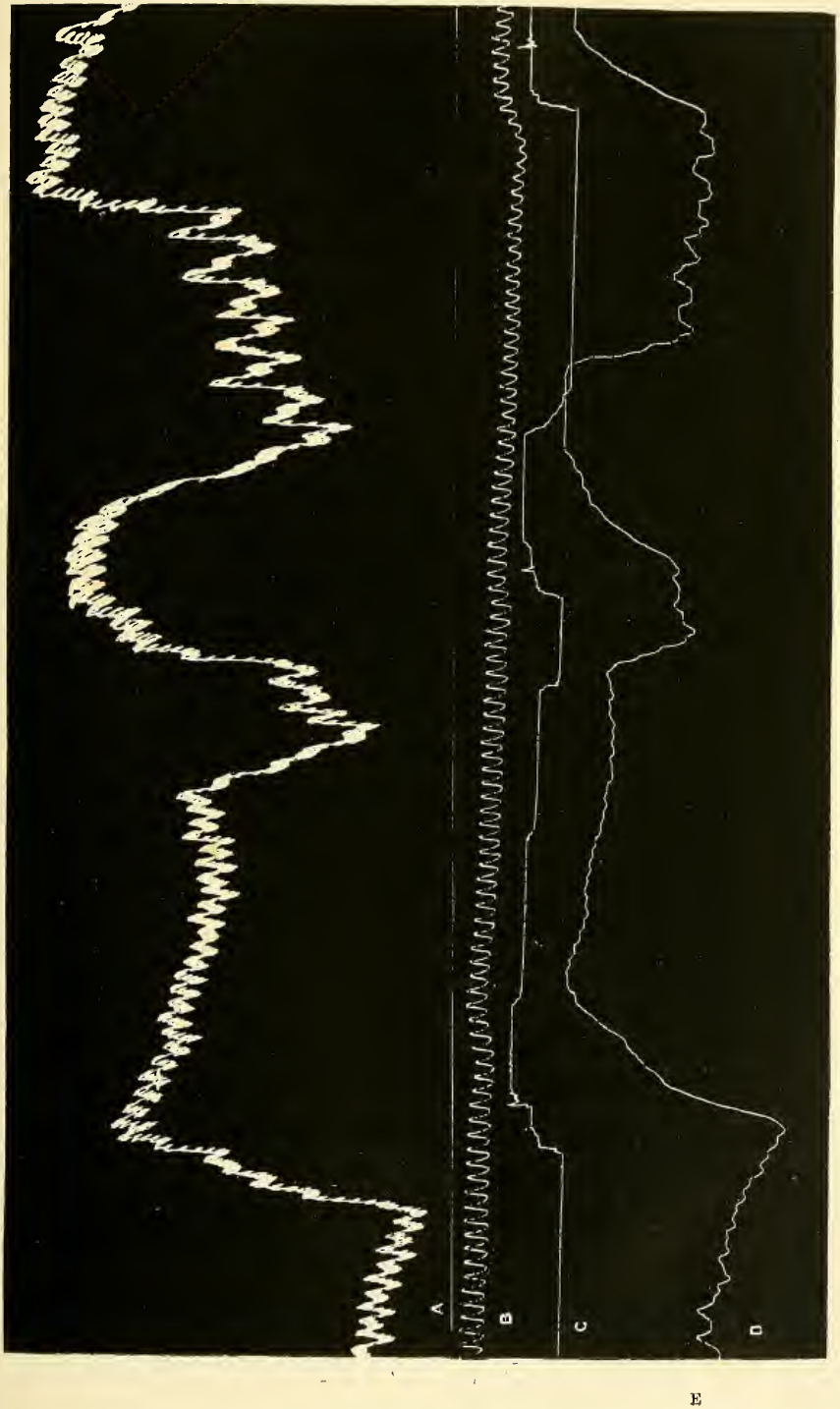



FIG. 11.

A. Carotid. B. Right Auricle. C. Intracranial Pressure. D. Torcular.

### SUPRARENAL EXTRACT.

Oliver and Schäfer have demonstrated the great and direct effect which an extract of the medulla of the suprarenal gland has on the arterioles. On the suggestion of Professor Schäfer, Bayliss and I employed the suprarenal extract in a final effort to obtain constriction of the cerebral vessel. On injecting suprarenal extract the arterial pressure rises greatly, the general venous pressure also rises, and the cerebral venous pressure passively follows the rise in the general pressures. In order to eliminate the overwhelming effect of the splanchnic constriction we attempted to tie the aorta and inferior vena cava, and so restrict the circulation to the upper part of the body. In ten successive experiments ligaturing the arch of the aorta just beyond the left subclavian artery has proved fatal in the course of a few minutes, although the vena cava inferior was tied immediately afterwards. The heart passed into delirium cordis. This, I believe, is due to the fact that the extensibility of the arch of the aorta is insufficient to allow the heart to empty itself. The intraventricular pressure therefore rises and cardiac anæmia is produced. In four animals we have ligatured the aorta just above the diaphragm and also the vena cava inferior, and the result has not been a fatal one. In these four experiments we have been unable to obtain any decided evidence of constriction in the cerebral vessels. On injecting the extract the arterial rise is very much smaller, but the cerebral venous pressure still passively follows this rise.

### EFFECT OF COLD.

On applying a freezing mixture to the head of a dog a slight rise of arterial pressure occurs, due to local vascular constriction. The cerebral circulation remains practically unaltered. On placing the cold mixture on the abdomen the rise of arterial pressure is greater, and the cerebral venous pressure follows this rise. Wertheimer has obtained the same result. It would therefore appear that the application of ice-bags to the head as a form of treatment has practically no effect upon the cerebral circulation.

Such being the experimental results, it now remains for me to attempt to answer the questions which at the beginning of this section I set myself to solve.

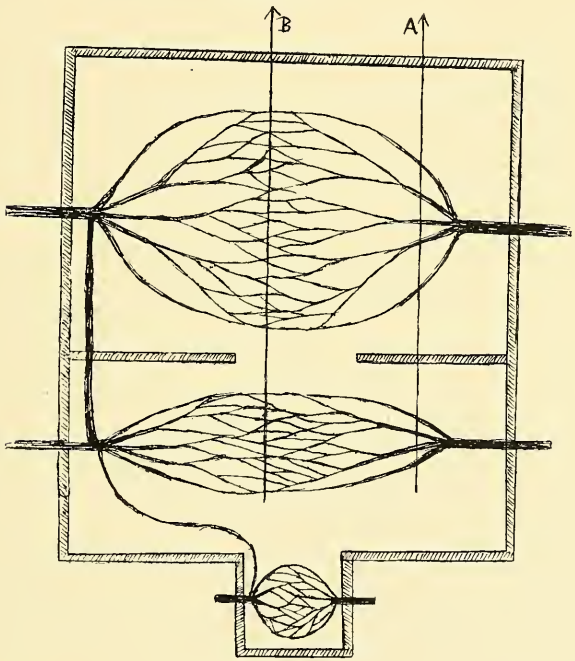
### THE PRINCIPLES OF THE CEREBRAL CIRCULATION.

I find that the volume of the brain is in the closed cranium almost an invariable quantity, and agree with the Monro-Kellie doctrine. When the arterial pressure rises, the expansion of cerebral volume can take place only to a certain limited amount. For as soon as all the cerebro-spinal fluid has been driven out from the cranium the brain is everywhere in contact with the rigid wall of the skull. The cerebro-spinal fluid in the cranium of the living animal is insignificant in amount. Any further expansion of the arteries and capillaries can now only take place by an equivalent compression of veins, for the semi-fluid brain matter is incompressible. The reservoirs of blood in the veins will therefore be so far constricted until the cerebral

venous pressure again becomes equal to the intracranial pressure; that is, to the pressure of the brain against the veins. Then the whole circulatory system of the brain will have assimilated itself to a scheme of rigid tubes. Thus the velocity of the blood-flow will be increased, and a relative distribution of the blood in the arteries, capillaries and veins will be changed. A rise of arterial pressure does not by any means, through compression of the cerebral capillaries and veins, produce an anæmia of the brain, but rather it causes an increased velocity of the current. Gaertner and Wagner found that this was so in experiments on the outflow of blood from the lateral sinuses, and their results have been lately confirmed by Nabarrow and myself in a research on the exchange of blood gases in the brain. We found that during the great rise of arterial pressure produced by absinthe the outflow of blood from the torcular increased from twice to six times. In this research the metabolism of the brain was found to be very little as compared with that of the muscles. It was Grashey who enunciated the doctrine that intracranial pressure depends in origin on the tension of the cerebral arteries, and that consequently a high pressure in these arteries must lead to decrease in the amount of blood flowing through the brain. Geigel went so far as to declare that every expansion of the cerebral arteries causes an anæmia and every constriction a hyperæmia of the brain. This view cannot be accepted. The intracranial pressure has been found by us in all physiological conditions to be the same as the cerebral venous pressure. It does

not depend directly on the tension of the cerebral arteries, but is an expression of the tension of the cerebral capillaries and veins. The intracranial pressure is, in fact, that tension which remains after the force of the heart has been expended in driving the blood through the cerebral arterioles. It is therefore

FIG. 12.




the same as the pressure in the venous sinuses. If the intracranial pressure becomes pathologically greater than the pressure in the venous sinuses, then the cerebral circulation is disordered. By no physiological means can intracranial pressure be *maintained* higher than cerebral venous pressure. It is, however, possible that a very sudden and abnormally high rise of arterial pressure should so expand the arteries at the base of

the brain as to temporarily express capillary areas and produce anæmia. Nevertheless, the effect can only be momentary, and then the circulation once more rights itself and the blood-flow becomes increased in velocity. This is understood by considering the following points (Fig. 12):

In Fig. 12 the circulation in the cerebral chamber, the cerebellar chamber, and the vertebral canal is diagrammatically represented with the anterior spinal and vertebral anastomoses. The whole box is, it is supposed, filled with a viscous, incompressible substance.

(1) The brain lies in a rigid box, and is incompressible.

(2) A sudden rise of arterial pressure by expansion of the cerebral arteries obliterates cerebral capillaries and veins (lines B and A, Fig. 12).

(3) The pressure that causes this obliteration is the arterial pressure minus the elastic tension of the arterial wall, and this pressure is transmitted directly through the brain substance.

(4) Directly the capillaries and veins are obliterated the pressure rises in them to the static pressure; that is, to the mean arterial pressure.

(5) The veins and capillaries, therefore, again become patent, because arterial pressure transmitted directly is obviously greater than arterial pressure minus the tension of the arterial wall transmitted through the brain substance.

(6) The expansion of the cerebral vessels thus reaches their utmost limit in the cranium, and they become so long as the high arterial tension lasts of the

nature of rigid tubes. By no possible means, therefore, can the circulation of the brain strangle itself in normal conditions, as has been supposed by many theorisers. Experimentally, except in the temporary way I have described, it is never found to do so. Haig has lately ascribed the production of certain cases of headache, epilepsy, and mental depression to high blood tension occasioned by the uric acid diathesis. He accepted Grashey's doctrine that high arterial pressure by compression of the cerebral veins produced congestion of the brain; such a pathological doctrine is, I believe, erroneous. Intracranial tension is, as I have shown in all the above experiments, variable in quantity. It is the same as cerebral venous pressure, and varies in the same direction absolutely as general venous pressure, and proportionately as general arterial pressure. It is of a purely circulatory origin. By intracranial tension I mean the pressure which the surface of the brain is exerting against the walls of the cranium. Cerebral pressure is an expression which has an old pathological significance, and in dealing with physiological facts it is necessary to avoid its use. The normal intracranial tension has been recorded by several observers. Leyden arrived at it by a troublesome but perfectly accurate method. He found the intracranial tension to equal about 100 mm. $H_2O$, and that it fell to zero when the carotids were divided and the animal bled to death. Bergmann estimated the tension of cerebro-spinal fluid at 80 mm. $H_2O$; W. Koch at 60 mm. $H_2O$; Key and Retzius at 156 to 260 mm. $H_2O$; Falkenheim and Naunyn at 100 to

150 mm. $H_2O$; Cybulski at 72 to 190 mm. $H_2O$. Quincke in two cases of meningocele recorded pressures of 15 and 52 mm. $H_2O$ respectively. Falkenheim and Naunyn, on occluding the abdominal aorta and thus raising the arterial pressure, found that the cerebro-spinal fluid increased its tension, but soon fell again owing to resorption of fluid. On increasing general venous pressure by distending a bag in the right auricle, they found that the tension of cerebro-spinal fluid rose to 200 mm. $H_2O$. I have, I think, sufficiently proved to what a degree the intracranial tension is an ever-varying quantity. This variation is the cause of the discordant results obtained by these authors. Once more to recapitulate: my method in researches on this matter has been to measure the intracranial tension by the brain pressure gauge, the cerebro-spinal fluid pressure by the means of a tube passed through the lamina of the atlas and connected with a manometer; and simultaneously I have made measurements of cerebral venous, general venous, and general arterial pressures. It is absolutely essential in these experiments that, in carrying out the operative procedure, no vessel within the cranial cavity should be wounded. A subdural hæmorrhage would, of course, make the readings much too high. The absolute correspondence of intracranial and cerebral venous pressure has been obvious throughout the experiments on cerebral circulation. I have shown that cerebro-spinal fluid pressure does not always absolutely correspond, since the brain can expand to a far greater degree than the spinal cord, and the brain on expan-

sion expresses the cerebro-spinal fluid from the cranium and comes into contact with the cranial wall. On the other hand, cerebro-spinal fluid cannot only distend the vertebral ligaments, but can leak away from the subarachnoidal space. Under the influence of gravity the intracranial or cerebral venous pressure may fall below zero. In strychnine spasms they may rise to 50 mm. Hg. In normal conditions, with the animal horizontal, the pressures generally are about 100 to 130 mm. $H_2O$. Cramer reckoned the cerebral venous pressure at 87 to 140 mm. $H_2O$; Key and Retzius at 70 to 210 mm. $H_2O$. My experiments prove that there is no compensatory mechanism by which the brain matter can be protected from great changes of circulatory pressure. Men remain conscious in the spasms of strychnine when the intracranial pressure must be equal to some 50 mm. Hg. On the other hand, I found that intracranial pressure was slightly below zero in a certain patient when standing upright. This patient had been trephined for symptoms of lunacy. It is therefore clear that the functions of the brain can continue at any pressure varying from zero to 50 mm. Hg. Bayliss and I have been unable to find any evidence of the local vaso-motor mechanism which Roy and Sherrington sought to establish on the grounds of experimental results which followed in asphyxia, and after injection of acids and brain extract. The cerebral circulation passively follows every change in the general circulation. Every change in the position of an animal, from the influence of gravity on the vascular system, affects the cerebral circulation.

Every variation in respiration and every muscular movement is followed by passive changes in the circulation of the brain. Every heart-beat and every respiratory undulation is exhibited on the tracings of the cerebral venous or intracranial pressures. Compression of the jugulars or the abdomen causes a marked rise in cerebral venous pressure, and muscular movements of the neck by pressure on the jugular veins are sufficient to affect the cerebral circulation. Every stimulus that enters the organism and affects the general vaso-motor centre produces a passive effect on the cerebral circulation. Each pleasurable emotion raises the general blood pressure and increases the blood-flow through the brain, and each painful emotion brings about the opposite result. It is by means of the splanchnic area that the blood supply to the brain is controlled. The overwhelming importance of the splanchnic mechanism in maintaining the circulation and life I shall show in the next section dealing with the influence of gravity on circulation. An anæmia of the central nervous system excites the vaso-motor centre, and if the splanchnic vessels constrict the blood pressure rises and more blood is driven through the brain. The same result is produced by asphyxia. We have in the vaso-motor centre a protective mechanism by which blood can be drawn at need from the abdomen and supplied to the brain. At the moment that excitation from the outside world demands cerebral response, the splanchnic area constricts and more blood is driven through the brain. On the other hand, after the taking of food the abdominal vessels dilate, the circulation is

slow in the brain, and the organism is unwilling to respond. The brain has no direct vaso-motor mechanism, but its blood supply can be controlled indirectly by the vaso-motor centre acting on the splanchnic area. The vaso-motor centre is part of the central nervous system, and feels the same needs and is stimulated by the same centripetal impulses as affect the rest of that system, and thus it maintains a supply of blood to the central nervous system which corresponds to its functional activity. There is undoubtedly muscular tissue in the vessels of the pia mater, and it may well be asked why is there muscle present if it does not constrict? Gulland has entirely failed to demonstrate vaso-motor nerves in the vessels of the pia. The muscle is an elastic supporting structure capable of withstanding internal tension, but as far as the principles of the cerebral circulation are in question it may be otherwise neglected. If there is constriction or dilatation of the cerebral vessels, it is so small that it is overcome passively by any rise in the general pressures.

## CEREBRAL CONGESTION.

On the grounds of experiment, arterial congestion of the brain can only be produced passively either by over-action of the heart or by the constriction of vascular areas in other regions of the body. Arterial hyperæmia of the brain may be the sequence, not the cause, of disturbances in the central nervous system. In animals this form of congestion seems to produce experimentally no symptoms, and I doubt the existence of

such a pathological state. It is to be particularly remembered that the vascular condition of the face and head does not necessarily correspond with that of the brain. Passive venous congestion, on the other hand, is shown by experiment to be of great pathological importance. The absolute relation of the cerebral circulation to the general venous circulation has been proved, and will be still further shown in the next section on the influence of gravity on the circulation. Any increase of general venous pressure will, by impeding the flow of blood through the brain, tend to produce anæmia of this organ.

## SUMMARY OF THE SECTION ON THE CEREBRAL CIRCULATION.

(1) No evidence has been found of the existence of cerebral vaso-motor nerves: either by means of stimulation of the vaso-motor centre, or central end of the spinal cord after division of the cord in the upper dorsal region: or by stimulation of the stellate ganglia, and, that is to say, the whole sympathetic supply to the carotid and vertebral arteries.

(2) Evidence is not forthcoming of the existence of any local vaso-motor mechanism.

(3) In every experimental condition the cerebral circulation passively follows the changes in the general arterial and venous pressures. The intracranial or cerebral venous pressure varies directly and absolutely with general venous pressure, but only proportionately with general arterial pressure.

(4) The intracranial pressure is in all physiological conditions the same as the cerebral venous pressure.

(5) The volume of the blood in the brain is in all physiological conditions but slightly variable.

(6) There is no compensatory mechanism by which the intracranial pressure is kept constant. The intracranial pressure or cerebral tension, which in all physiological conditions is circulatory in origin, may vary with the circulatory pressure from zero to 50 mm. Hg. The functions of the brain matter continue in this varying condition of pressure.

(7) In all physiological conditions a rise of arterial pressure accelerates the flow of blood through the brain and a fall slackens it. The cerebral circulation is controlled by the vaso-motor centre acting on the splanchnic area.

(8) There is no evidence of the causation of cerebral anæmia by spasm of the cerebral arterioles.

(9) Arterial hyperæmia of the brain produces no experimental results of importance. Cerebral venous congestion, on the other hand, is of great pathological significance.

## SECTION IV.

### THE INFLUENCE OF THE FORCE OF GRAVITY ON THE CIRCULATION OF THE BLOOD.

The influence of the force of gravity on the circulation is a question of very obvious importance, yet it is one curiously neglected by physiologists. The physician and the surgeon daily observe the effect of the position of the body upon the rate of the pulse, the sounds of the heart, diseases of the heart and lungs, hypostatic pneumonia, varicocele, erectile tumours, piles, varicose veins. The conditions of shock, anæmia, hæmorrhage, chloroform poisoning, are also seen to be largely affected by the position of the body. But on turning to the text-books of physiology nothing is to be found within their pages upon so important a theme, and on seeking still further into the physiological journals and archives few and imperfect are the researches there chronicled.

One of the earliest observers was Piorry, the distinguished French physician. He insisted on separating cerebral syncope from cardiac syncope. "In cerebral syncope," he writes, "the heart continues to beat, but the beats have not force enough to overcome the resistance which is given by gravity." Therefore the

activity of the brain is suspended. If the force of gravity is made to aid the heart in propelling blood to the brain the syncope will cease. Piorry observed several cases bearing on this point. In one instance he was suddenly called to a patient who had lost consciousness. The respiration was rare and stertorous, sensibility was abolished, the pulse very feeble, the heart very weak and irregular, and the face pale. The patient had been supported by his friends in the sitting posture for fifteen minutes. Piorry refused to bleed the patient and laid him down horizontally. Immediately his eyes opened, respiration was accelerated, the colour came back to the face, and in three minutes all the unfavourable symptoms had disappeared.

One such case arose from hæmorrhage, another from emotional effect produced by a slight operation. Piorry was summoned to another patient who had been trampled upon in the belly by a horse. He was found supported by his friends in the sitting posture and almost dead. Piorry immediately placed him horizontally, and the patient at once recovered. After Piorry's departure the patient insisted on taking his seat again in his carriage. On doing so he immediately became unconscious and died.

Piorry further investigated the problem by experimenting on dogs. He placed them in the vertical feet-down position, and bled them from the jugular vein till syncope was induced. By placing them in the vertical feet-up position the animals were at once restored to consciousness. And every time he

repeated this experiment of alternating the positions he obtained the same result. "It is in consequence of gravity," writes Piorry, "that when the arms are held down, the veins swell and the capillaries are filled, and that the reverse occurs when the arms are held up, that varicoceles enlarge on standing up, and diminish in the horizontal position, and that the head and face redden when held down. It is in consequence of gravity that pneumonia invades the posterior border of the lung when the patient lies on his back, and for the same reason, if the patient for a length of time be laid upon his face, the pneumonia appears in the anterior border of the lung."

Piorry concludes that the force of gravity has a very marked effect on the circulation, and that this is especially observable in patients who are weakened by any cause. He affirms also that the effect of altering the position of a patient will determine the diagnosis between apoplexy and syncope.

Marshall Hall, in a research on the effects of loss of blood, found that after recovery from a severe hæmorrhage the syncopal condition could be at once reinduced in dogs by placing them in the vertical feet-down position, then "the countenance and eye languish, the head droops, the mouth opens, the respiration is panting, and the heart is scarcely to be heard." Immediate relief was given by reversing the position.

Salathé, Cybulski, and others have shown experimentally that the arterial pressure in the carotid falls

in the vertical feet-down position, and rises in the vertical feet-up position.

Salathé and Mendel have recorded that centrifugal force acts in the same way. When the animal is centrifuged in the feet-out position, the influence of the centrifugal force causes the blood pressure to fall in the carotid artery.

Salathé, Brissaud, and Franck have noticed the aspirating effect of the vertical feet-down position on the brain in man.

Two pupils of Hermann, Blumberg, and Wagner have fallen into fallacies concerning the influence of gravity on the circulation, owing to the attempted application of the principle of a known artificial schema to the unknown and living circulatory system. For fuller particulars of these researches I must refer to my paper on this subject (*Journal of Physiology*, 1895).

My attention was first drawn to the influence of gravity upon the circulation by observations which I made upon the normal intracranial tension in a patient of Dr. Claye Shaw. The patient had been trephined, and Dr. Shaw asked me if I could estimate his normal intracranial pressure. I did so by an adaptation of the principle of my brain pressure gauge.

The trephine hole was protected by the scalp. To a cast of the patient's skull I had moulded a glass apparatus which (as shown in Fig. 13) consisted of—

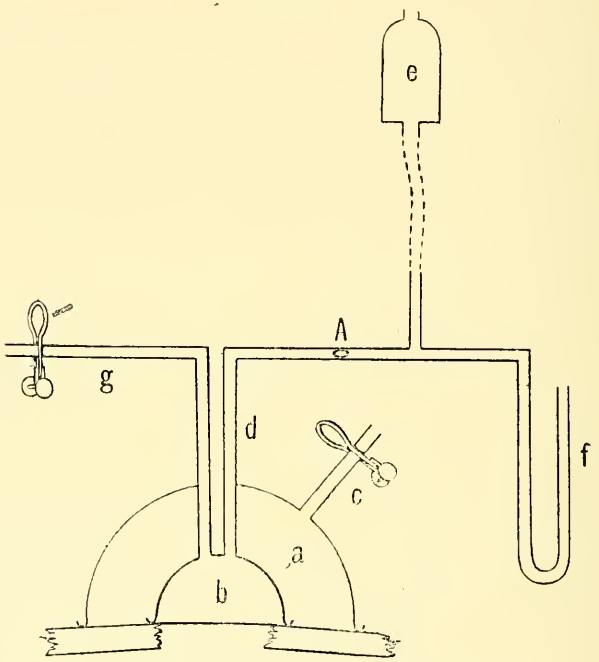
(a) An outside cupping-glass which could be aspirated by a mercury pump through the tube (c).

(*b*) An inside chamber which fitted to the edge of the trephine hole. From this inner chamber passed two tubes:

i. A tube (d) leading to a pressure bottle (e) and to a manometer (f).

ii. A tube (g) closed by a clip.

FIG. 13.




The skull, previously shaved, was well vaselined, the apparatus applied, and the outside chamber aspirated. An air-tight joint between the inner and outer chambers was thus obtained around the trephine hole. The inner chamber was then filled with water from the pressure bottle by opening the clip on the tube (g). The clip was next closed and the pressure bottle lowered till the water was on a level with the

trephine hole. The tube (d) was then clipped for a moment, and an air-bubble introduced at the point (A). The clip being opened, any alteration of pressure could be observed by the position of the air-bubble, and measured by altering the pressure bottle and bringing the air-bubble back to its initial position at zero pressure. I found that the pressure was negative while the man sat upright, but that it became positive as soon as the head was bent down towards the knees or on any expiratory effort. The air-bubble exhibited large cardiac and respiratory undulations. Experimenting on dogs, I found that exactly the same thing occurred. The normal cerebral pressure became markedly negative in the feet-down posture, and positive in the feet-up posture. For the further investigation of the subject I constructed an animal holder, which could be swung round a horizontal axis. In the axis—and this is essential*—the cannula connected with the vessel under observation was always placed; the cannula was itself connected with a fixed mercury manometer.

The chief results of this research I will now show in a series of tracings. I shall use the term hydrostatic moment to express the simple passive change produced by the hydrostatic effect of gravity, dynamic moment to express the changes of the cardiac vasomotor and respiratory mechanisms; and these, as I shall show, compensate for the hydrostatic moment. The first is a purely physical effect, the second a vital phenomenon. The experiments were con-

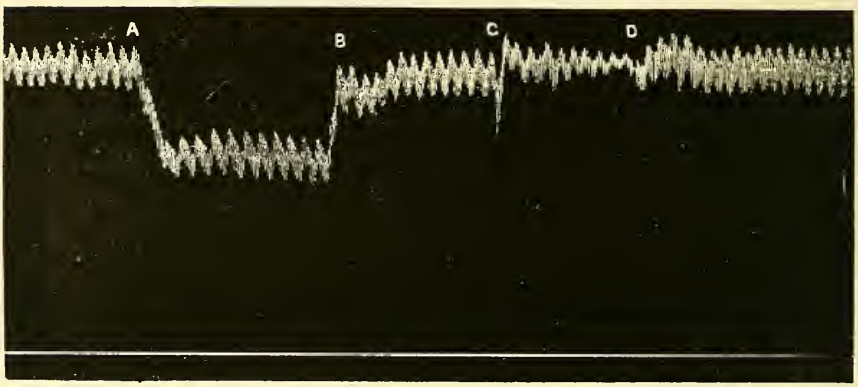
* Ref. *Journal of Physiology*, 1895, pp. 18–21.

ducted on rabbits, dogs, cats, and monkeys, in the state of morphia narcosis. In certain experiments the influence of other anæsthetics was investigated:

I. NORMAL EFFECT.

Fig. 14. Fox-terrier. Carotid artery in axis.
A. Vertical, feet down. C. Vertical, feet up.
B. Horizontal. D. Horizontal.

FIG. 14.



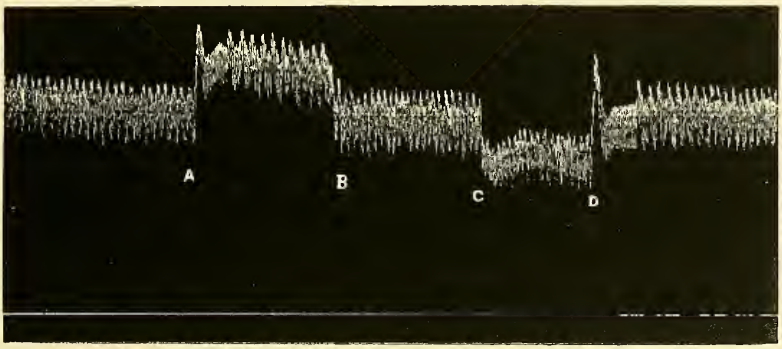

On placing the animal in the vertical feet-down position the pressure is seen to fall rapidly, and to remain permanently lowered. On restoring the animal to the horizontal position the pressure rises with equal rapidity to almost the original normal level. This rapid rise of pressure occasions a slight and compensatory fall, followed by the absolutely normal condition.

On the vertical feet-up position being taken, momentary cardiac inhibition occurs with a fall in pressure, then a permanent rise of pressure follows;

and on returning the animal to the horizontal position there is first a fall of pressure, then a slight compensatory rise, and lastly the normal pressure is again restored. The greater effect is seen to be produced by the feet-down posture.

On rapidly alternating the positions, I have found that the blood pressure rises and falls with each change of movement.

FIG. 15.




The feet-up position accelerates respiration, the feet-down position slows it. These effects are reversed by dividing the vagi.

Fig. 15. From the same Fox-terrier as Fig. 14. Femoral artery in axis.

A. Vertical, feet down. C. Vertical, feet up.
B. Horizontal. D. Horizontal.

The effects are now shown to be exactly the reverse of those shown in the carotid trace. These two tracings, therefore, show the simple hydrostatic effects which I have found to be the same in all animals examined, although they are more or less masked in

different individuals owing to compensatory changes or dynamic effects.

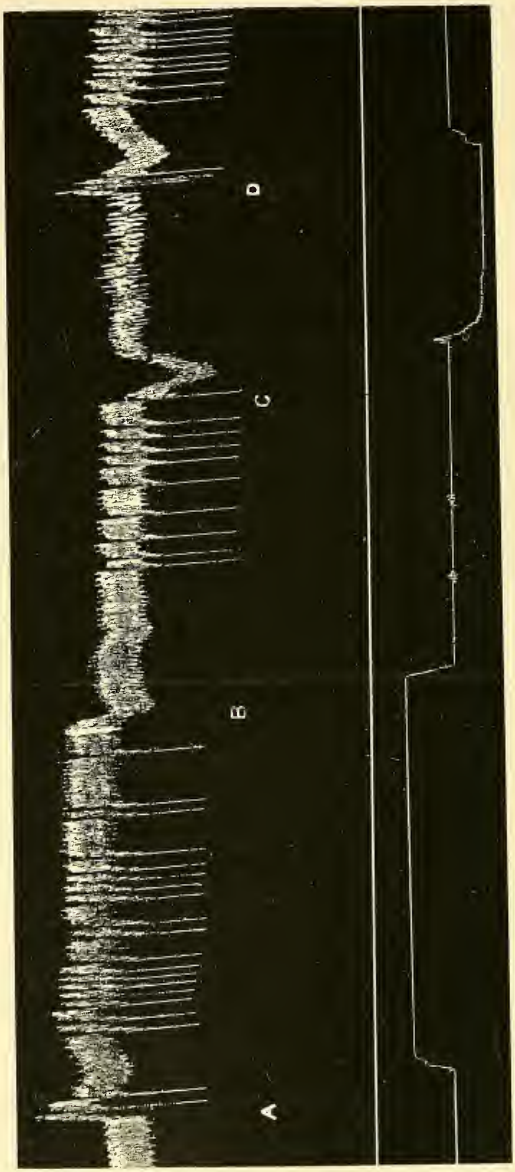



Fig. 16.

Fig. 16. Fox-terrier. The upper line shows the carotid trace.

The lower line is a record taken by the brain pressure gauge. The axis passed close to the trephine hole in which this apparatus was screwed, so that a slight correction is necessary for the changes of blood pressure, since the carotid cannula was not in the axis.

A. Vertical, feet up.
B. Horizontal.
C. Vertical, feet down.
D. Horizontal.

I found in this dog that the hydrostatic effects were almost completely compensated for in the arterial system by the dynamic effects. Cardiac inhibition is found in the feet-up position, acceleration in the feet-down position. This compensatory inhibition is abolished by section of the vagi. The intracranial pressure rises about 16 mm. Hg in the feet-up posture, and falls about 16 mm. Hg in the feet-down position. With this effect the tracing of cerebral venous pressure is to be compared.

FIG. 17.


Fig. 17. Monkey (*Macacus sinicus*). Vagi divided.

A. Vertical, feet down.
B. Horizontal.
C. Vertical, feet up.
D. Horizontal.

This tracing shows that over-compensation can take place in a monkey under morphia narcosis. The tracing was of exactly the same character before the vagi were divided.

On placing the animal with the feet down the pressure rises, and on changing to the horizontal position the over-compensation is still more plainly to be seen. At that moment, while the vessels of the animal are still in a state of compensatory constriction, the rise of pressure due to the hydrostatic moment takes place, and there is therefore a large rise. As the constriction passes off the normal level is again reached. In the feet-up position an over-compensatory dilatation is recorded. The hydrostatic moment of gravity is in the same direction in all the animals—rabbits, cats, dogs, monkeys. It is the dynamic moment that differs. The compensation with individual differences is incomplete in nearly all rabbits and dogs, but in monkeys it is far more complete. There is, however, a striking difference in individual monkeys. The bonnet monkey of the present tracing over-compensated, while neither a *Macacus rhesus* nor a *Ceropithicus callitrichus* monkey compensated fully for either position. The question of compensation, as will be seen later, is greatly affected by the anæsthetics, but these individual differences have been studied under the same uniformity of morphia narcosis.

The effect of gravity on the venous circulation was determined in (1) the splenic vein, and (2) the femoral vein.

(1) The cannula in the splenic vein was in connection with a fine-bored manometer filled with saturated $MgSO_4$ sol. This manometer was placed in connection with a delicate piston recorder. The pressure in the splenic artery was recorded at the same time. Both cannulæ were placed in the axis.

On placing the animal feet downwards the pressure in the splenic artery scarcely varied, but the form and rate of the respiratory undulations were markedly different.

On the other hand, the pressure in the splenic vein —that is to say, the portal venous pressure—rose over 100 mm. $MgSO_4$ sol., while the respiratory undulations became very marked. The large rise of portal pressure in the feet-down position, as will be noticed later, was caused by the determination of blood to the veins of the splanchnic area. In consequence of this there should be a fall on the arterial side, but under the influence of gravity the compensatory constriction in the arterioles of the splanchnic area prevented a more marked fall of pressure from being shown in the splenic artery.

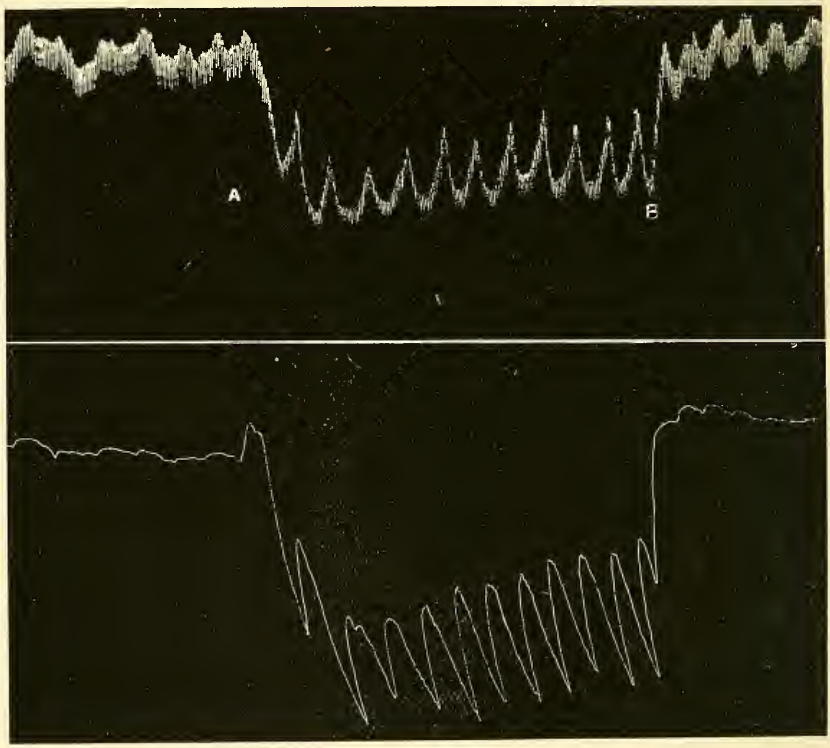
(2) The pressure was recorded in the central end of the femoral vein, the valves having been destroyed by passing a stylet up into the vena cava. The vein was connected with the $MgSO_4$ manometer, and this itself was in connection with a delicate air tambour. The pressure in the femoral artery was simultaneously recorded. Both cannulæ were placed in the axis.

Upon placing the dog with the feet down a rise in arterial pressure took place, and there was also a

marked change in the character of the respiratory undulations. The rise in the vein was equal to 200 mm. of $MgSO_4$ sat. sol.

On turning the animal feet upwards the reverse

FIG. 18.




effect was given and the venous pressure became negative. (For these tracings see *Journal of Physiology*, 1895.)

Fig. 18. Fox-terrier. The carotid pressure and the cerebral venous pressure are recorded in this tracing. The axis passed near the torcular, so that a correction

is necessary for the carotid, the recorded fall of pressure being slightly too little.

A. Feet up. B. Horizontal.

On turning the animal with the feet down the cerebral venous pressure fell more than 100 mm. of sat. $MgSO_4$ sol. below the zero pressure. The respiratory undulations are shown to be extremely exaggerated on both curves. On placing the animal feet upwards the reverse result was given. The effect on the cerebral venous pressure can be compared with the effect on intracranial pressure recorded in Fig. 16. Both methods give the same results. Intracranial pressure is always the same as the cerebral venous pressure.

When the animal's head is raised above the trunk the influence of gravity can be at once observed by opening the torcular. The pressure is then negative and no blood flows out, but on depressing the head the blood at once begins to flow.

The reverse of these same phenomena can be observed in the central end of the femoral vein. No blood will flow from the cannula in the feet-up position, but when the animal is placed with the feet down the blood flows out freely.

From the above facts it is manifest that with the skull intact and compensation for the hydrostatic effect incomplete the circulation stagnates in the brain in the feet-down posture, and with the skull opened, if the cerebral capillary blood pressure falls below that of a column of blood reaching from the heart to the brain, the capillaries will empty and the brain collapse. This collapse of the brain can be experimentally

observed by trephining the skull when the animal is

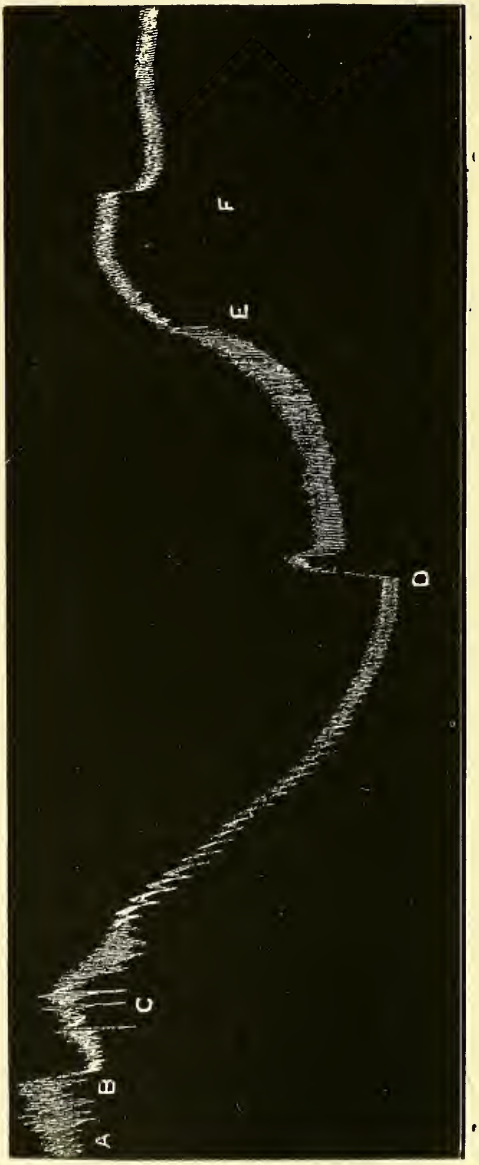



FIG. 19.

in the feet-down position and the blood pressure is very low, and the same thing was noticeable in the

patient of Dr. Claye Shaw; whenever this man stood upright, the scalp over the trephine hole was sucked in.

Fig. 19. Fox-terrier. Anæsthetic, ether and chloroform in equal proportions. Carotid in axis.

A. Moderate anæsthesia.
B. Feet down.
C. Anæsthetic pushed.
D. Feet up.
E. Anæsthesia still complete.
F. Horizontal.

The effect on the influence of gravity of pushing this anæsthetic is shown in this tracing. On placing the animal with the feet down and putting it under moderate anæsthesia with a mixture of ether and chloroform the fall was slight. On rapidly pushing the anæsthetic the pressure ran down quickly towards the zero line. At the point D, immediately after the withdrawal of the anæsthetic, the animal was turned from feet down to feet up. The pressure rose considerably at once, and then more gradually till the animal in a condition of anæsthesia was once more placed in the horizontal position. This tracing shows that the fall of pressure was due partly to vaso-motor paralysis and partly to cardiac failure. The blood could be driven by gravity to the heart from the veins, but the heart could not pass it on to the arteries.

Figure 20. Fox-terrier. Carotid in axis. Anæsthetic, chloroform.

A. Feet down.
B. Chloroform pushed.
C. Chloroform removed.
D. Abdomen compressed.
E. Compression removed.
F. Feet up.

This tracing shows that the splanchnic area is the

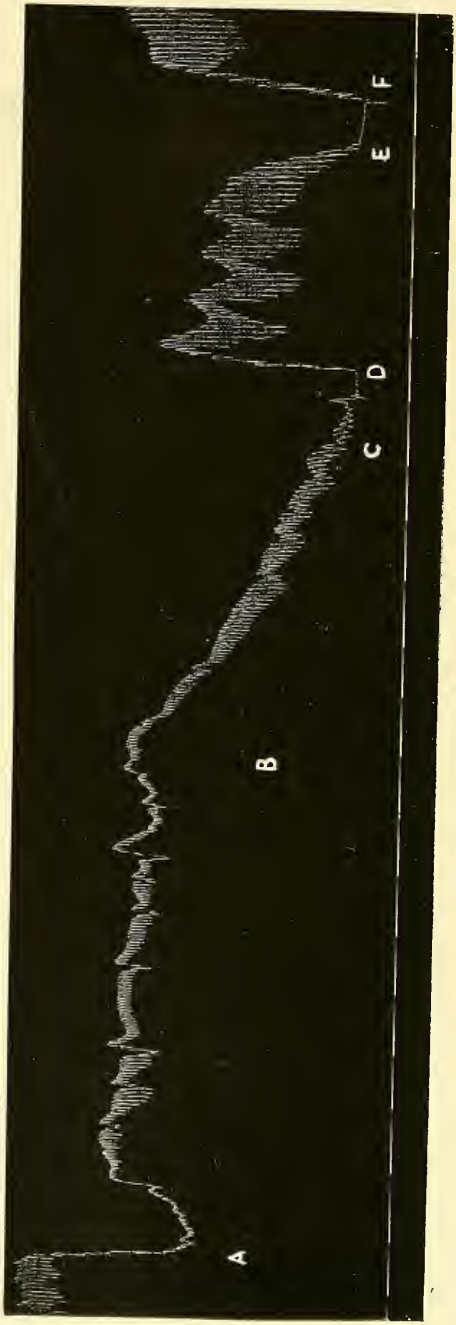



FIG. 20.

seat of the vaso-motor paralysis induced by the anæsthetic.

By firmly compressing the abdomen at a time when the pressure was rapidly sinking to zero, recovery was brought about. The pressure fell again immediately on removing the compression from the abdomen, to be once more restored by the feet-up position. It is noticeable, however, that the feet-up position did not restore the pressure to a higher level than that which was maintained in the feet-down position before the anæsthesia was pushed.*

The effect of chloroform on monkeys is the same, and is uninfluenced by the maintenance of artificial respiration throughout the administration of the anæsthetic.

Fig 21. Fox-terrier. Carotid in axis. Anæsthetic, ether.

A. Feet down and anæsthetic pushed.
B. Anæsthetic withdrawn. D. Compression removed.
C. Abdomen compressed. E. Horizontal.

The striking contrast between the slow fall produced by pushing ether and the rapid fall produced by chloroform in the feet-down position is shown in this tracing. In moderate amounts, ether does not interfere with the compensation, and when this anæsthetic is pushed the fall is very gradual. A third of the tracing was removed in order to reduce the size for reproduction, the fall therefore is still more gradual than is exhibited. Finally, most of the compensation was abolished, although the heart-beats remained

* The larger heart-beats after D are chiefly due to the dislodgment of a clot in the cannula.

strong. The abdomen was then compressed and the

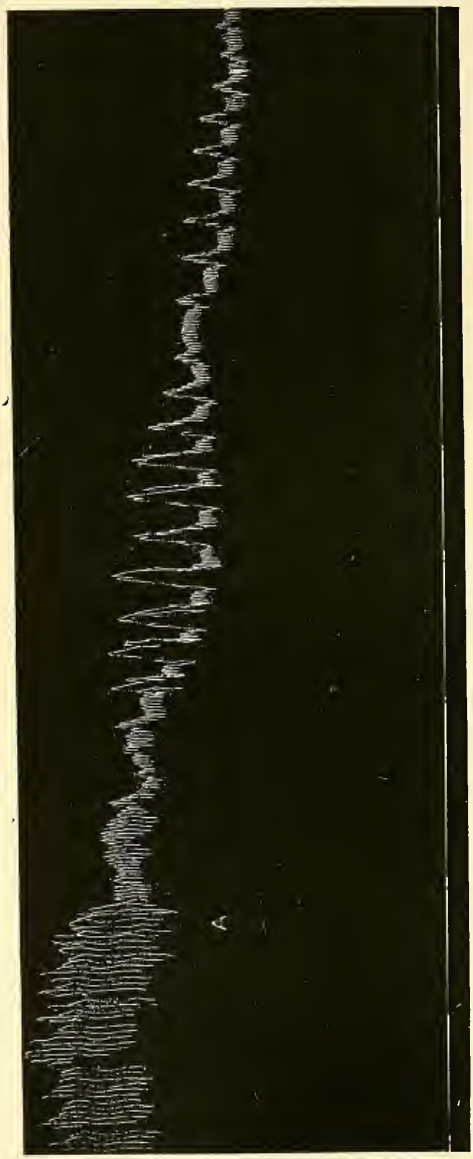


Fig. 21A.

blood pressure was immediately restored. On removing the compression the pressure fell again, and

was finally restored by placing the animal in the horizontal position.

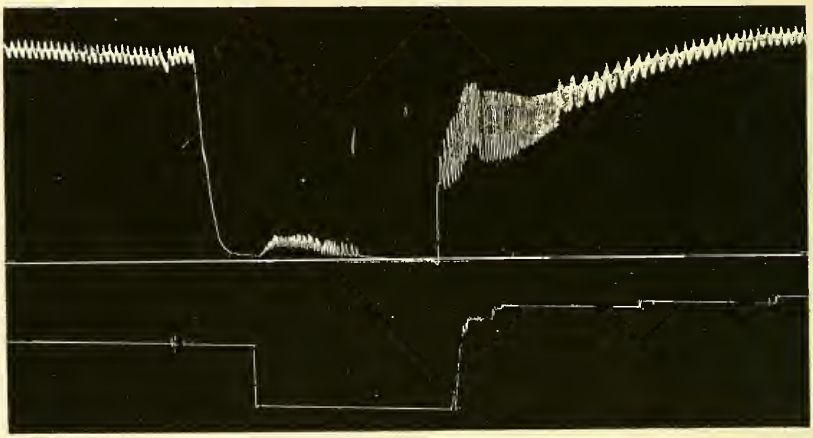

FIG. 21B.

Both ether and chloroform when pushed, damage the compensation for some time after these anæsthetics have been removed.

Fig. 22. Fox-terrier. The spinal cord was divided between the sixth and seventh dorsal vertebræ and destroyed with a stylet as far as the third dorsal vertebra. The carotid and intracranial pressures are recorded. The axis passed close to the trephine hole.

The correction in reference to the axis, for the

FIG. 22.

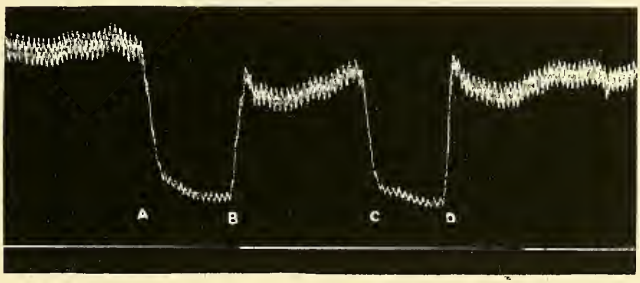


carotid pressure made necessary in passing from the horizontal to feet-down position, is slight enough to be ignored. After the cord was divided in the upper dorsal region the carotid pressure in the feet-down position is seen to fall to the zero line. Section of both splanchnic nerves produces the same results, likewise the injection of such drugs as curari or amyl nitrite, which paralyse the vaso-motor mechanism. The heart-beats which succeed the primary fall were occasioned by violent respirations. These respirations are of a peculiar gasping type. The abdomen is

maintained in the retracted position and deep thoracic inspirations are made. The retraction of the abdomen forces blood up from the veins of the splanchnic area, while the thoracic inspirations exert a suction action.

The total effect of each respiration was to draw some blood into the right heart and occasion a heart-beat. Exhaustion soon followed, caused no doubt by the anæmia of the respiratory centre. This anæmia is shown by the fall in intracranial pressure, by the

Fig. 23.




cessation of respiration and of the heart-beats, and by the apparent death of the animal. If, in such an experiment, the thorax is opened and the heart observed, the right side of the heart is seen to be completely empty when the animal is placed in the feet-down position, but it continues to rhythmically contract.

The instantaneous recovery brought about by the feet-up position is shown. The blood is actually shot out of the vena cava and from the veins of the splanchnic area into the heart.

From the tracings of the vena cava and the splenic vein I have proved that the blood passes into the

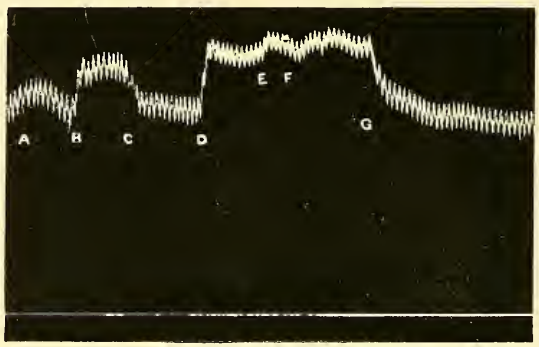
abdomen. This is also shown by the effect of bandaging the abdomen.

Figure 23. Cat. Anæsthesia moderate. Ether and chloroform in equal proportions. Carotid in axis.

A. Feet down.
B. Broad bandage drawn round abdomen.
C. Bandage removed.
D. Horizontal.

The tracing exhibits the effect of bandaging the

FIG. 24.




abdomen. In the condition of ordinary anæsthesia the animal had little power of compensation; this could, however, be artificially supplied to it, for as long as the bandage was maintained the pressure remained normal. It is possible, by firmly strapping, to drive the pressure up beyond what is even the normal level in the horizontal position.

Fig. 24. From the same cat.

A. Horizontal.
B. Abdomen bandaged.
C. Bandage removed.
D. Feet up.
E. Abdomen bandaged.
F. Bandage removed.
G. Horizontal.

In this tracing is shown the slight effect of bandaging the abdomen in the horizontal position, and the insignificant effect in the feet-up position in contrast to the enormous effect in the feet-down position.

In another experiment I determined the effect of bandaging the abdomen in a still more significant way.

The dog was placed in the horizontal position with the carotid in axis, the abdomen firmly strapped, and the spinal cord divided in the upper dorsal region. On placing the animal feet downwards the blood pressure fell only a few millimetres. After a few minutes I removed the strapping from the abdomen, the pressure immediately fell to zero, and the animal died. If the wall of the abdomen is very freely divided by a crucial incision when the animal is in the feet-down position, the blood pressure will fall largely in consequence of the withdrawal of mechanical support from the splanchnic vessels.

I was interested in finding an observation recorded by Stephen Hales in his classical researches on blood pressure: "When the blood has subsided a little in the tubes which were fixed to the arteries of these dogs, it would, as in the horse's, rise on a sudden considerably on deep sighing, as also on pressing the dogs' bellies hard with the hand the blood would immediately rise about six inches, and subside as much on taking off the hand; and it was the same on several repetitions."

Roy and Adami found that compression of the abdomen increased the quantity of blood thrown out

by the heart, to the extent of 26 per cent. (or even more) during the period of compression.

Excitation of the peripheral end of the divided spinal cord, of the splanchnic nerves, asphyxia or injection of suprarenal extract will equally well restore the blood pressure in the feet-down position by constricting the splanchnic area.

In one experiment I compressed the abdomen when the animal was in the later stage of asphyxia.

The bandage was applied at the point when the pressure was just beginning to fall, and the result was an immediate rise of pressure and great acceleration of the heart, produced by the sudden access of a large quantity of blood. This was followed by sudden failure of the heart and the fall of the blood pressure to zero. On supplying it with artificial respiration, the animal rapidly recovered, and the effect of re-bandaging the abdomen after recovery was very slight. (For this and other tracings see *Journal of Physiology*, 1895.)

This experiment shows the danger of throwing too much work on the heart when in an impoverished state. The same thing may occur in chloroform poisoning.

### DISCUSSION OF THE EXPERIMENTAL RESULTS.

The great importance of the position of the body in the methods of treatment employed in medicine and surgery is confirmed by these experiments. They, above all, point to the necessity in cases of syncope, shock, hæmorrhage, and chloroform poisoning, of main-

taining the cerebral circulation and filling the heart by elevating or strapping the abdomen.

That the influence of gravity is brought to bear in ordinary emotional syncope is clear from the success of lowering the patient's head between the knees. Syncope is commonly regarded as being due to a cessation of the action of the heart. It is, however, well known that in numberless cases of syncope the heart does not cease to beat, but continues to pulsate rapidly and very feebly, although no sign of the radial pulse can be felt.

It is commonly stated, however, that syncope consists essentially in sudden failure of the action of the heart. Now, no nerve is known which can produce such an effect except the vagus nerve, and we have not the slightest ground for assigning this action to that nerve. If the vagus nerve were excited, the heart would cease to beat or beat slowly, not rapidly or feebly.

Another view put forward is that in most cases fainting must be regarded as the influence of emotions upon the centres of the vaso-constrictor nerves of the cerebral hemispheres. This theory cannot be supported, for it has been impossible to determine the existence of any cerebral vaso-constrictor nerves.

The symptoms of syncope are exactly similar to those conditions observed in an animal placed feet downwards with the power of compensation destroyed by the production of vaso-motor paralysis. The empty heart continues to beat rapidly and feebly, but the pulse cannot be felt, the cerebral circulation

ceases, and hence consciousness is abolished. A deep sigh is the first obvious sign of improvement. A sigh is a deep thoracic respiration, with retraction of the abdomen. The compensatory effect produced by such a respiration is observable in many of the tracings. The animal or the patient in the condition of syncope is immediately restored on being placed in the horizontal or feet-up posture.

It therefore seems legitimate to suggest that ordinary emotional syncope is produced by sudden and temporary inhibition of the vaso-motor centre, caused by some painful and powerful sensory stimulation. Asthenic individuals with the least power of compensation would be most prone to syncope. The evolutionary etiology of syncope, if I may enter so far on the path of speculation, possibly lies in the fact that danger is avoided by the sudden fall into the horizontal position and the simulation of death. This theory of syncope can be easily tested on patients by the results obtained on firmly pressing or strapping the abdomen. The customary practice of placing the patient's head down between the knees to remove faintness points to the truth of this view.

The power of compensation is probably largely affected by the habits of the individual animals. Gerdy pointed out that vinedressers become, for instance, able to work all day in a bending position, with the head down.

Salathé suggested that patients who have lain long in bed lose the power of adapting themselves to change of position, and become like quadrupeds, and

hence the faintness, dizziness, and danger of syncope which occur during convalescence when the patient first rises from bed.

Dr. George Oliver has investigated the effects of position on the diameter of the radial artery in man, by means of his most ingenious instrument, the arteriometer. Many of his results obtained by an entirely independent method agree with the results obtained by me on monkeys. Dr. Oliver finds that in normal men the radial diameter is largest in the vertical position ; that is to say, the effect of gravity is over-compensated for. On the other hand, when asthenia is the predominant clinical condition, the radial calibre in the standing posture is often remarkably reduced.

The first day I returned to the laboratory after a severe attack of influenza, Dr. Oliver found the diameter of my radial artery to be 1·6 mm. sitting upright, and 1·9 mm. recumbent. One week later the diameter was 1·9 sitting, and 1·55 recumbent, and I had completely recovered my compensatory power, and had lost all feeling of faintness on rapidly changing my position.

On the first day compensation was supplied to me by compression of my abdomen, and the diameter of my radial was driven up from 1·6 mm. sitting to 1·9 mm. (sitting) by this means.

I find that the arteriometer affords an accurate method of *recording* in suitable cases from day to day the power of compensation in changes of position, and therefore of the condition of the vaso-motor mechanism, and the value of the lines of treatment pursued.

*Compensation for gravity is the clinical key to the condition of the vaso-motor mechanism.*

The significance of these experiments in regard to shock cannot be doubted. After death from shock or syncope the abdominal veins, on post-mortem examination, are commonly found to be engorged with blood. "The story told by the symptoms of shock is one of depression of all the vital functions associated with the evidence of a diminished circulation of blood in those portions of the periphery which we can examine during life. The integument is blanched and shrunken, the pulse is thready or imperceptible, the veins are collapsed, and open wounds, unless involving large arterial trunks, bleed slightly or cease to bleed."

These symptoms are exactly exhibited by an animal which, after the production of vaso-motor paralysis in the splanchnic area, is placed feet downwards. Since Goltz's experiments of paralysing the splanchnic vessels in the frog by sharply striking the abdomen, splanchnic paralysis has been the commonly received explanation of the pathology of shock. Many of the after effects of shock can be associated with the consequent cerebral anæmia. In a case of shock after operation, when the patient remained in a befogged mental condition, Dr. Oliver found that the radial diameter was half what it became when the patient recovered.

The cause of shock may, I believe, be sought entirely in vaso-motor paralysis, either central or peripheral. A state of shock primarily arises from the fall of blood pressure, and consequent anæmia of the central

nervous system. Secondarily, death results when the arterial pressure becomes insufficient to drive the blood to the respiratory centre. Such a state of shock can be rapidly produced in animals by one method only, and that is—with the aid of gravity—by complete splanchnic paralysis. This paralysis can be produced rapidly by—

(1) Complete section of the abdominal wall and exposure of all the viscera, so that they may depend freely from the wound and the vessels become paralysed by exposure.

(2) Section of the cervical spinal cord or splanchnic nerves, or destruction of the vaso-motor centre by the knife, or its paralysis by poisons.

(3) By local vaso-motor poisons, such as chloroform or amyl nitrite, pushed to excess.

The state of shock is not rapidly produced in carefully anæsthetised animals by any other means. Laurie and the Hyderabad Commission tried but entirely failed to produce shock in animals by evulsion of teeth, of nails, crushing of spermatic cord, &c. They expected immediate results.

The condition of shock, on the other hand, always advances slowly during the course of a prolonged experiment on an animal. The blood pressure slowly falls, and the rate of this failure of the vaso-motor mechanism depends on the primary strength of the animal, on the severity of the operative interference, and largely on the position of the animal and the anæsthetic employed.

When this state of shock exists, the respiratory

and the vaso-motor centres are failing on account of the cerebral anæmia, and Cheyne Stokes respiration, and Traübe Hering curves of blood pressure become common.

In this state, and even after cessation of respiration, compression of the abdomen will raise the blood pressure, and restore the respiratory centre to activity.

It seems possible therefore that bandaging or elevating the abdomen may be a treatment of some value in cases of shock, and especially where an operation is urgently required.

The influence of position after severe hæmorrhage was long ago experimentally proved by Marshall Hall. From the present research it seems evident that it is the abdomen that must be elevated and bandaged; this treatment applied to the limbs only, as is the usual practice, can be of far less value. Neither can the injection of normal saline be of much benefit, for as long as the splanchnic floodgates be open wide the salt solution will simply collect in the all-devouring abdominal veins. The rapidity with which normal saline, without causing more than a very small and only temporary rise of the arterial pressure, can be driven into the circulation is a fact well known. The injection of saline, on the other hand, causes a marked and far more permanent rise in venous pressure.

That the danger of administering chloroform is increased in the vertical position, or with the head raised above the trunk, is obvious from the results recorded in this section. It is possible that death may

result from vaso-motor paralysis although the empty heart is continuing to beat. The result is entirely opposed to the conclusion of the Hyderabad Commission.

On examining the tracings published by this Commission, Mr. Wall and I have found that in every case the blood pressure falls enormously before respiration ceases. The failure of respiration in the Commission's experiments is as obviously due to that anæmia of the respiratory centre which is produced secondarily by the fall of blood pressure, as it is in all my own experiments. The fact that the primary and vital danger is failure of cerebral circulation stares one in the face in every one of the tracings made by this Commission.

Undoubtedly respiration generally stops before the heart, because if the head is above the level of the heart the cerebral circulation will cease before the circulation in the coronary arteries. Gaskell and Shore by their beautiful cross circulation experiments have conclusively proven that chloroform can paralyse the heart when the central nervous system is entirely cut off from the drug and respiration continues. This paralysis not only affects the heart, but the blood vessels, as I have proved by my experiments.

Abdominal compression immediately removes the effect of the vaso-motor paralysis, and thus the administration of chloroform is absolutely safe during parturition owing to the tension within the abdomen.

It is important to remember that the vaso-motor paralysis continues for some little time after the removal of the anæsthetic, and on this ground, and

also by reason of the shock after any operation, it is indicated that the patient's abdomen should be elevated or compressed. The causes of death from chloroform are undoubtedly, as MacWilliam has said, three in number: (1) respiratory failure; (2) vaso-motor paralysis; (3) dilatation of the heart from poisoning of the cardiac muscle.

The first cause of danger can be immediately abolished by artificial respiration, the second by abdominal compression; the third cause is often irremediable. I have, however, frequently brought animals round by rhythmic compression in sequence, first of the abdomen, after of the thorax. The compression of the abdomen fills the heart with blood, the compression of the thorax drives some of this blood into the arterial system, and at the same time artificially respirates the animal. The effect of this procedure can be recorded by the arterial manometer. Compression of the abdomen also drives venous blood directly into the cranio-vertebral sinuses and so on into the cerebral capillaries, and thus, if the hæmaglobin be not entirely reduced, brings a certain supply of oxygen to the medullary centres. Very often natural respiratory movements can be kept up by this means for five or ten minutes, although the heart remain completely and hopelessly paralysed.

I would venture to strongly urge this method of relief in cases of chloroform syncope in man. Maintained compression of the abdomen or elevation of the trunk is dangerous, as the determination of a large quantity of venous blood to the heart may only increase its paralytic dilatation The abdomen and thorax

should be firmly, *alternately*, and rhythmically compressed.

In the light of these experiments the existence of an open trephine hole or the removal of large pieces of the cranium must be seen to have a most important effect on the cerebral circulation. As long as the skull is closed the cerebral vessels cannot empty under the influence of the hydrostatic moment, even if the compensation be weakened or abolished; that is, unless cerebro-spinal fluid be secreted into the intracranial cavity to a corresponding amount, and this is negatived by the experiments which I have carried out on the subject. So soon, however, as the skull is opened and atmospheric pressure is brought to bear upon the brain, the cerebral capillaries will immediately collapse, and the blood be withdrawn from the brain whenever the cerebral capillary pressure falls below that of a column of blood, the height of which is measured from the heart to the brain.

This explains the rapid death which can be sometimes induced in animals by placing them feet downwards after trephining the skull, as Regnard and Salathé have found.

The position selected by patients suffering from lung or heart disease can be explained, for it is manifest that the upright posture will afford the greatest relief, by diminishing pulmonary congestion through the retention of a large quantity of blood in the splanchnic area.

The common practice of wearing abdominal belts for weakness, and the application of binders after parturition, find a physiological explanation in the

results of this research. Roy and Adami have suggested that the almost universal practice of wearing waist-belts and stays is due to the fact that compression of the abdomen increases the output of the heart and the blood supply to the brain and muscles.

As, however, I have shown that the normal monkey compensates perfectly for changes of position, and Dr. George Oliver has by measurements with the arteriometer found the same thing in normal healthy men and women, the practice of tight-lacing cannot be held necessary on physiological grounds, and probably owes its origin entirely to the instinct of sexual attraction.

Cases are occasionally recorded of patients, some of whom lose their memory in the standing position, and regain it in recumbency; others can only do mental work when in the horizontal position, and others suddenly are at a loss for memory when attempting to address a public meeting.

The Rabelaisian effects of fear which are so commonly manifested by men before battles, examinations, public performances, &c., the vomiting, diarrhœa and involuntary micturition, and the loss of memory more rarely met with, may all be associated with temporary splanchnic dilatation and determination of venous blood to the abdomen. A loop of intestine, properly fed with arterial blood, remains altogether at rest; if the blood become venous vigorous movements begin. In such cases a tight abdominal belt might be of considerable service, as in any other condition in which the normal compensation for gravity is abolished.

## CONCLUSIONS.

To briefly summarise: there is little difficulty in interpreting these experiments and in drawing the following conclusions from them:

(1) That the force of gravity must be regarded as a cardinal factor in dealing with the circulation of the blood.

(2) That the important duty of compensating for the simple hydrostatic effects of gravity in changes of position must be ascribed to the splanchnic vaso-motor mechanism.

(3) That the effects of changing the position afford a most delicate test of the condition of the vaso-motor mechanism.

(4) That the amount of compensation depends largely on individual differences.

(5) That the compensation is far more complete in upright animals, such as the monkey, than in rabbits or dogs, and therefore is probably far more complete in man.

(6) That, in some normal monkeys, over-compensation for the hydrostatic effect occurs.

(7) That in the normal monkey and man gravity exerts but little disturbing influence, owing to the perfection of the compensatory mechanism.

(8) That when the power of compensation is damaged by paralysis of the splanchnic vaso-constrictors, induced by severe operative procedures or by injuries to the spinal cord, the last stages of asphyxia, or by some

poison such as chloroform, then the influence of gravity becomes of vital importance.

(9) That the feet-down position is of far greater moment than the feet-up position, because when the power of compensation is destroyed the blood drains into the abdominal veins, the heart empties, and the cerebral circulation ceases.

(10) That, generally speaking, the feet-up position occasions no ill consequence.

(11) That the horizontal and feet-up positions at once abolish syncope induced by the feet-down posture, by causing the force of gravity to act in the same sense as the heart, and thus the cerebral circulation is renewed.

(12) That firmly bandaging the abdomen has the same effect (while the heart remains normal, and so long as the mechanical pressure is applied to the abdominal veins, the pressure cannot fall).

(13) That if the heart is affected by chloroform poisoning the restoration of pressure is incomplete, and it is possible that the heart may be stopped altogether by the inrush of a large quantity of blood, caused by too rapid application of pressure on the abdomen (more work would be thrown on the heart than in its impoverished condition it could perform).

(14) That vagus inhibition and cardiac acceleration are subsidiary compensatory mechanisms in the feet-up and feet-down positions respectively.

(15) That chloroform rapidly paralyses the compensatory vaso-motor mechanism and damages the heart.

(16) That ether, on the other hand, only paralyses the compensatory vaso-motor mechanism slowly and when pushed in large amounts. The partial asphyxia produced by ether helps to maintain splanchnic constriction.

(17) That the vaso-motor paralysis induced by these anæsthetics lasts for some considerable time after the removal of the anæsthetics.

(18) That chloroform can, by destroying the compensation for gravity, kill the animal if it be placed with the abdomen on a lower level than the heart.

(19) That elevation or compression of the abdomen immediately compensates for the vaso-motor paralysis produced by chloroform.

(20) That rhythmic alternate compression of the abdomen and thorax is the best means of restoring an animal from the condition of chloroform collapse.

(21) That the feet-down position inhibits respiration, and the feet-up accelerates it.

(22) That these respiratory results probably depend upon the stimulation of sensory nerve-endings by changes of tension brought about by the alterations of position, because the results are abolished by dividing the vagi.

(23) That in the feet-down position the respiration is thoracic in type, and the abdomen is retracted; in the feet-up position the respiration is diaphragmatic and the abdomen freely expanded.

(24) That these types of respiration tend to compensate for the effects of gravity on the circulation, for the retraction of the abdomen in the feet-down

position mechanically supports the abdominal veins, whilst the thoracic inspirations aspirate blood into the heart. In the feet-up position the full and free expansion of the abdomen withdraws all obstacles to the compensatory dilatation of the abdominal veins.

## SECTION V.

### CEREBRAL ANÆMIA.

The effect of ligature of the carotid arteries has been known from the earliest days. The very name carotid betokens the word sleep, and among the Greeks these arteries were called soporales. Peripatetic mountebanks used to include a goat among their stage properties, and were said to tie up and relax these arteries in the animal so that at their pleasure the goat fell down motionless and stupid, or at their bidding leapt up again with great vigour. It was a custom among the Assyrians to compress these arteries in their young men before circumcision so that they might feel the less pain. John Bell is most contemptuous of these traditions. He writes: "Valsalva, Van Swieten, Pechlinus, Lower, and especially Drelincurtius, spent days and weeks in tying up the carotids of dogs. What does all this imply? Surely a strong belief in tales which would disgrace the 'Arabian Nights.' Tales concerning the manner of tying the cord round the neck of a she-goat, or even of a young man, so that without hurting them they should be made to sleep or wake according to the bidding of the spectators." "Morgagni," he adds, "talks more

seriously of the she-goat and of this snibbing of the young men of Assyria than one could wish in respect of the character of one so truly great as Morgagni." The first important experiment on this subject was that of Astley Cooper. He tied both carotids in a rabbit and compressed the vertebral arteries. Spasms immediately resulted and the respiration ceased. In the dog, Cooper ligatured the four cerebral arteries. The animal recovered after a preliminary stage of paralysis and lived to be an excellent house-dog. Panum attempted to repeat this experiment, but tied the vertebrals at the spot where they pass over the atlas and after important branches have been given off to the anterior spinal artery. Panum explained Cooper's successful result by anastomosis through these branches, but, as is shown by the original preparation in the Museum of the College of Surgeons, Cooper tied the vertebrals at their origin from the innominate and subclavian arteries. In a more recent research, Kussmaul and Tenner tied the left subclavian and the innominate arteries in rabbits suddenly and without opening the pleura. Spasms occurred in from ten to forty-five seconds. The immediate symptoms were loss of consciousness and voluntary movement. These were followed by clonic spasms beginning in the muscles of the neck. Then occurred dilatation of the pupils and cessation of respiration save for respiratory gasps at long intervals. These respiratory gasps continued for three to five minutes after the application of the ligatures. After complete occlusion of these arteries for not longer than two to three minutes, the

brain showed the power of complete recovery on the ligatures being once more loosened. The sudden re-entry of blood immediately stopped the spasms, and in no case did it cause them. It was anæmia only that produced spasm.

On loosening the ligatures the animal often gave a sudden jump forward, then remained paretic for a few moments, and finally recovered fully. They found no spasm occurred in weak or over-anæsthetised animals. Horses, goats, depend entirely upon the carotids for their cerebral blood supply, since the vertebrals where they enter the basilar are reduced to mere vascular threads. It has been recorded that occlusion of the carotids only is sufficient to produce spasms and loss of consciousness in these animals. So in spite of Bell's scoffs the mountebanks were in the right. Kussmaul and Tenner localised the seat of the spasms to the mid-brain and medulla oblongata. They could not obtain fits by ligaturing the cerebral veins, but succeeded by this means in slowing the respiration. Jolly showed that the Kussmaul-Tenner spasms were not caused by any sudden change of the intracranial tension such as is produced by cutting off the blood supply. He injected saline into the carotid at the ordinary arterial pressure, and thus washing out the blood produced the typical spasms. Hermann and Esher on ligature of the superior vena cava and azygos veins obtained no effect. This shows how free the cranio-vertebral venous anastomosis is. When these authors also blocked the veins of the spinal cord and vertebral canal by means of a cautery they obtained a typical spasm, occurring, however,

after a more prolonged interval. Thus, on ligaturing the four arteries the spasms appeared in ten seconds, on blocking all the veins in twenty seconds. Ferrari injected wax into the venous sinuses. He obtained no effect unless every venous sinus was blocked, when an epileptic fit occurred, with first a slowing then a final cessation of respiration. Among other workers on this subject Poisseuille and Magendie observed after the ligature of both carotids an acceleration of the pulse and a rise of blood pressure. Ehrman, Vulpian, and others noticed that the quickening of the pulse and respiration took place before the cessation of the latter in cases where the four cerebral arteries were ligatured. Similarly, Mosso obtained slowing of the pulse with a rise of blood pressure, and, on the other hand, acceleration of the pulse when the vagi were previously cut. Ehrman examined the brain through a glass window placed in the skull, and saw that on compressing the carotids the cortex slowly paled, but reddened again in a few moments. Coutey employed the method of embolism to obtain a complete arrest of the cerebral circulation. He curarised his animals and then injected lycopodium spores into the carotid. The arrest of the circulation of the whole brain caused cessation of respiration, an enormous rise of arterial pressure and slowing of pulse. The heart was at first sometimes completely inhibited. In some minutes death followed, preceded by acceleration of the heart and fall of pressure. Embolism of the hemispheres alone caused slowing of the pulse, with no rise of blood pressure. The greatest pressor effects occurred when the lower

part of the bulb was affected and the upper part of the cervical cord.

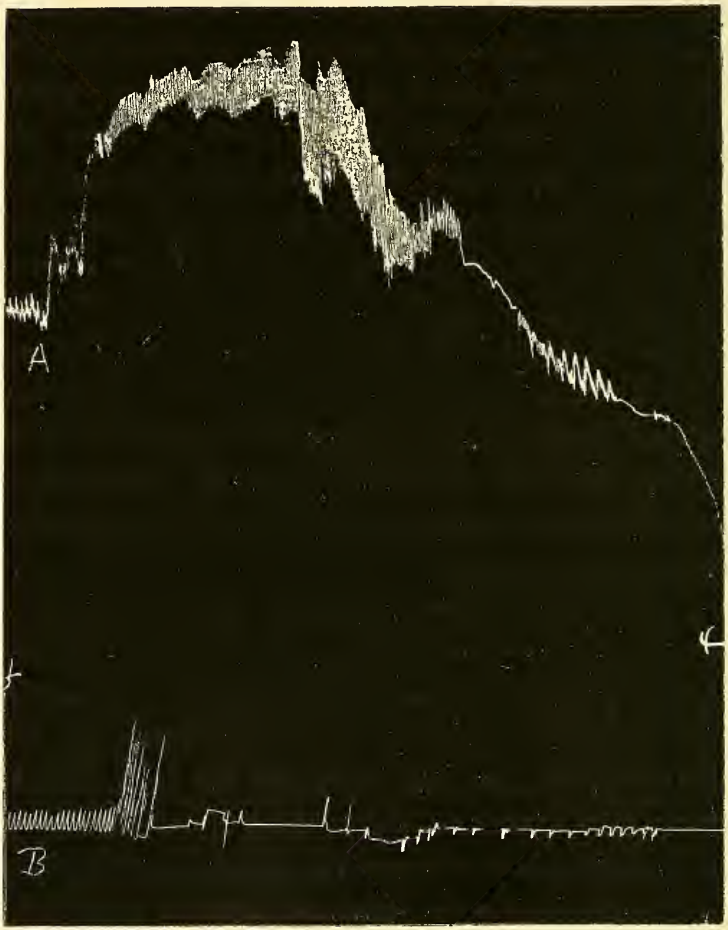
Bastgen injected oil into the peripheral carotid of goats, and produced a high blood pressure with slow pulse. Markwald injected wax into the peripheral carotid. The amount of embolism produced by these authors was determined by post-mortem examination. If the cerebrum was entirely cut off from the circulation, Markwald found that the animal was sleepy and unable to walk, while the limbs remained in abnormal positions. The animals did not respond to stimulation of light or sound. If the mid-brain was embolised, opisthotonos occurred; and if the vagi were divided, respiratory spasm of the diaphragm follows. If the basilar artery was embolised by injections into the vertebrals, the blood pressure rose; and after some spasms from failure of respiration, death quickly occurred.

I have many times ligatured the four cerebral arteries in different species of animals, confining myself to the pure experiment of tying the two carotids and the two vertebrals. If the innominate and subclavian arteries are tied—and this has been done by nearly all observers—the possibility of anastomosis by the superior intercostal and thyroid axis is destroyed. Nearly all rabbits die from the ligature of the four arteries exactly in the manner described by Astley Cooper and Kussmaul and Tenner. Spasms, rise of blood pressure, ineffectual respiratory gasps at long intervals, fall of blood pressure and death, form the sequence of symptoms as is shown in Fig. 25.

A. Carotid. B. Respiration.

If at the moment of ligature the blood pressure be low and the cerebral circulation feeble no spasms will

FIG. 25.




then occur. The blood pressure will not rise. There simply follows cessation of respiration and a fall of the blood pressure to zero. If artificial respiration be maintained, the circulation will then continue although

the blood pressure fall from paralysis of the vaso-motor centre.

Two rabbits out of the dozen or so that I have experimented upon have survived the quadruple ligature during the hour in which they were kept under observation. In these animals the blood pressure rose and remained at a higher level, while respiration was accelerated and deepened for a brief period before returning to the normal. On injecting into the arterial system of these animals a carmine gelatine mass, the medulla and mid-brain were found to be filled by way of the intercostal, anterior spinal, and basilar arteries. The injection did not, however, reach the upper surface of the cerebrum or cerebellum. When the quadruple ligature was applied to cats, about forty per cent. of these animals died in the same way as the rabbits; but in no dog of the many in which I have carried out this experiment did death ensue. In their case the blood pressure rose somewhat and remained higher, and the respiration (with one exception of a preliminary acceleration) continued unaffected.

On the post-mortem injection of these dogs a free anastomosis is found by way of a branch from the superior intercostal which passes in with the brachial plexus and joins the anterior spinal artery which becomes the basilar artery. From the other intercostals and deep cervical arteries there arise also anastomoses with the anterior spinal at each vertebral segment. When a record is taken of the cerebral venous pressure in the torcular Herophili and the four arteries ligatured, venous pressure is seen

at first to markedly fall and then to gradually recover.

Corin has found that dogs were not killed when all the four arteries were tied, and he could see by recording the pressure in the peripheral end of the carotid that the circle of Willis became filled with blood. In some cases, he says, the pressure in the peripheral carotid rose to a higher level than that at which it was before the ligatures were tied.

This paradoxical result of Corin's, if true, can only be explained by the conditions being such that the blood pressure was low before making the ligature, and that the pressor effect was very great after the ligature.

Hürthle obtained no evidence of collateral circulation in rabbits after ligature, by his record of the pressure in the peripheral carotid. On the other hand, Gaskell and Shore in their cross-circulation experiments on chloroform, found evidence of some circulation in the bulb in rabbits even after ligature of the innominate and left subclavian. In the case of three monkeys, when I have ligatured all four arteries the result has not been immediately fatal. One monkey was debilitated by the anæsthetic, and died some twenty minutes after making the ligature from vaso-motor paralysis. Another monkey survived during the period of observation (two hours), exhibiting a maintained rise of blood pressure with no alteration of respiration. The third monkey died at the end of six hours of vaso-motor paralysis.

I conclude, then, that the immediate effect of liga-

ture of cerebral arteries in different species of animals is as follows :

Horses and goats die from the effect of the ligature of the carotids only.

Immediate death occurs in the greater number of rabbits from ligature of the four cerebral arteries.

In the case of cats, one-third die and two-thirds survive.

Dogs all survive, and monkeys survive during the few hours they have been kept under observation.

The symptoms of cerebral anæmia are the same whether produced by ligature of arteries or by embolism. If the cerebrum be rendered bloodless, loss of consciousness and motor paresis arise. If the mid-brain and medulla are cut off from the circulation, spasms result ; vaso-motor spasm producing a high blood pressure, respiratory spasm, and a slow heart caused by spasm of the vagus centre. The state of spasm is followed by paralysis, as is shown by the cessation of respiration, fall of blood pressure, and quickening of the heart. Generally the primary cause of death is failure of respiration. If this be compensated for by artificial respiration the heart continues to beat, and then the vaso-motor paralysis which ensues may be a secondary cause of death. This can be compensated for by compression of the abdomen and placing in the vertical feet-up position. In some rare cases vaso-motor paralysis may be the primary cause of death.

I will illustrate these facts by the following tracings taken from cats.

In Fig. 26 is shown :

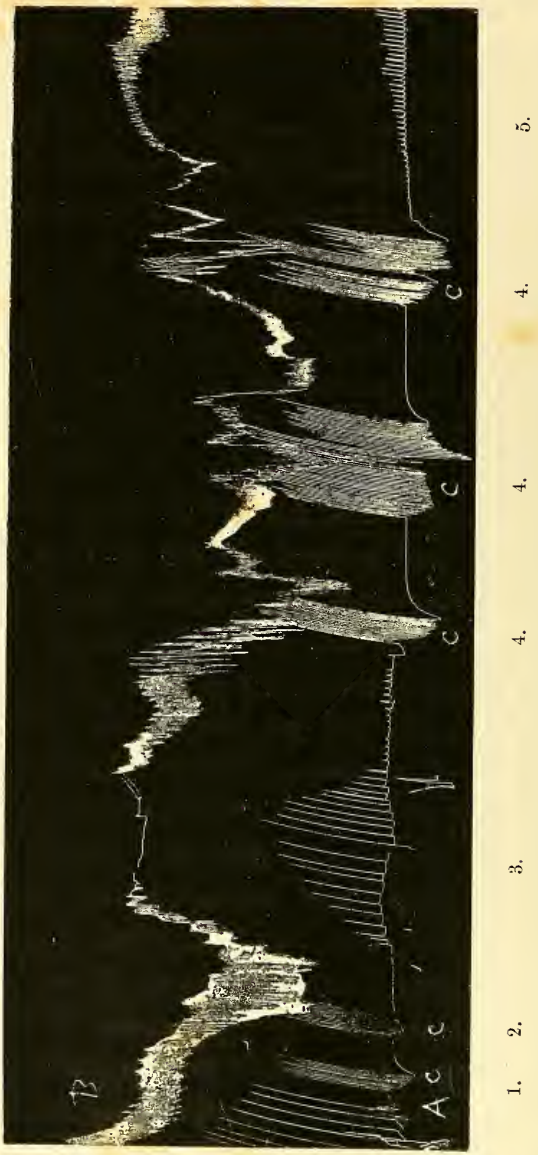



FIG. 26.

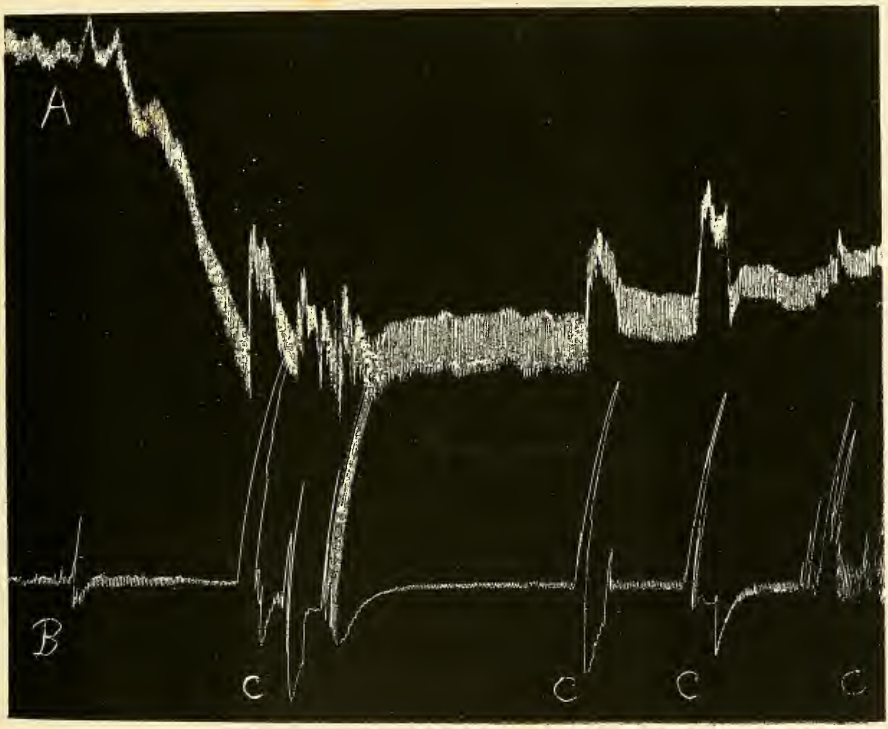
A. Respiration. B. Carotid.
C. Artificial respiration.

(1) The ineffectual spasms of respiration and fall of blood pressure caused by ligature of the four arteries.

(2) Recovery by artificial respiration, followed by asphyxial rise of blood pressure.

(3) Cheyne Stokes group of natural respiration,

Fig. 27.




followed again by cessation of respiration and fall of blood pressure.

(4) Recovery by artificial respiration and abdominal compression.

(5) Subsequent staircase respiration occasioned by the rise of blood pressure.

In Fig. 27 is shown the rarer failure of the vaso-

motor before the respiratory centre. Recovery was brought about by abdominal compression.

A. Carotid. B. Respiration.

C. Abdominal compression.

FIG. 28.


In Fig. 28 is shown :

(1) Ineffectual respiratory spasm.

(2) Vaso-motor paralysis and apparent death.

(3) Recovery by rhythmic alternate compression of the thorax and abdomen.

A. Carotid. B. Spasms of respiration.
C. Rhythmic compression.


Fig. 29.

Fig. 27 is exactly similar to a record taken of death resulting from chloroform. In these rarer cases of cerebral anæmia, as in chloroform poisoning, death is

caused by primary circulatory failure, and secondarily by respiratory failure. Alternate compression of the abdomen and the thorax may avert the fatal termination in both conditions.

On closing the last of the four arteries in an animal which survived in good condition the effect on the blood pressure was always pressor, the effect of again opening one artery depressor. This is shown very well in Fig. 29.

*Cat.*—A. One carotid opened. B. Closed.

(At the beginning of the tracing all the four arteries were closed.)

When the four arteries are closed in the cat the centres, in the medulla, in consequence of their partial anæmia, are in a very unstable condition. Thus, if a very little chloroform or $MgSO_4$ solution be allowed to enter the circulation, the centres are rapidly paralysed. This is shown in Fig. 30.

*Cat.*—Four arteries tied. Failure of respiration and fall of blood pressure caused by the entrance of a very little $MgSO_4$ solution while washing a clot out of the arterial cannula.

Recovery by artificial respiration. Subsequent staircase respiration.

A. Carotid. B. Respiration.

C. Artificial respiration.

Cheyne Stokes respiration and Traube Hering blood pressure curves are of common occurrence in these cases of cerebral anæmia. Chloroform and $MgSO_4$ solution bring them out with the greatest ease. Gad has produced them in rabbits by cerebral

anæmia. Gaskell and Shore have done likewise by a slight dose of chloroform coupled with cerebral anæmia. In normal animals large doses of morphia are

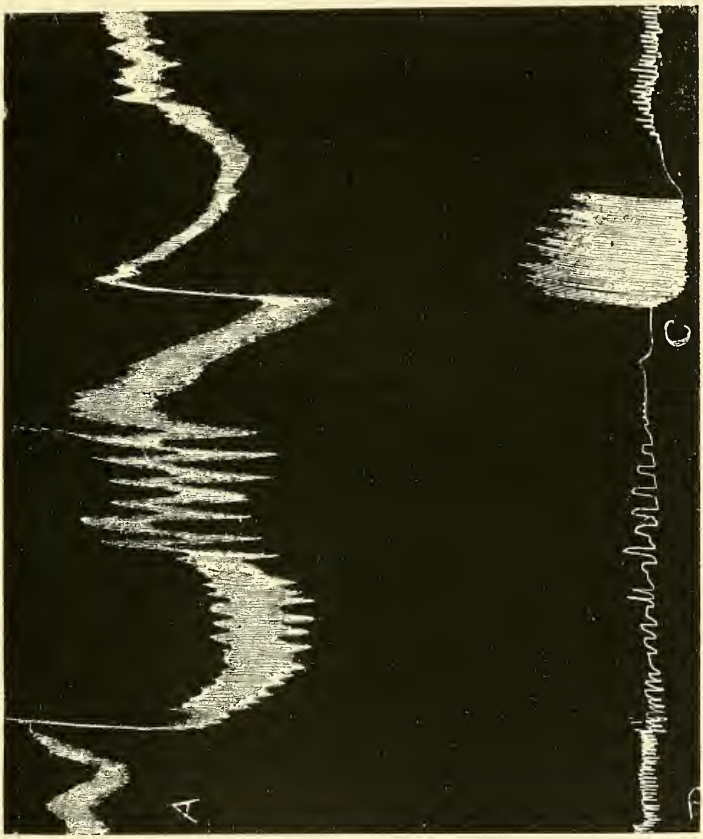



FIG. 30.

especially liable to produce these curious phenomena. It is always noticeable that as the blood pressure rises and falls in the Traube Hering curve so the respirations rise and fall in depth.

Filehne has shown in the case of Cheyne Stokes respiration, produced by morphia, that there is no

relation between the arterialisation of the blood and the rhythm. The group of respirations continue after the blood has become oxygenated and the pauses continue after the blood has become venous.

All my experiments on the cerebral circulation prove that a certain blood pressure is necessary to provoke respiration—that is to say, a certain amount of blood must flow through the centre. When from vaso-motor paralysis produced by chloroform or anæmia the circulation ceases in the medulla oblongata, respiration also ceases. If the blood pressure be again raised, the respiration begins in staircase fashion and dies away again as the blood pressure once more falls. In the condition of cerebral anæmia an asphyxial constriction of arterioles may raise the blood pressure and start a group of respirations. On arterialisation of the blood the blood pressure again falls, and consequently the respirations cease. Anæmia, morphia, or $MgSO_4$ solution may cause a rhythmic activity in the vaso-motor centre, producing Traube curves with a fairly high blood pressure. Respiration then occurs in Cheyne Stokes groups, for the respiratory centre is stimulated to greater efforts by each rise of blood pressure.

In the light of the results obtained by Filehne and Kionka, the Cheyne Stokes phenomenon must, I think, be regarded as similar to apnœa. The increased blood tension and blood flow provokes a group of increasing respirations. These are followed by, I believe, an apnœic pause similar to that pause produced by vagus excitation on forced ventilation of

the lungs. The respiratory centre is at the same time, through the action of anæmia or of the particular drug which calls out these phenomena, in a condition of lowered metabolism.

The theory of Filehne, that the group form of respiration depends on spasm of the vessels supplying the bulb, has no experimental support. He supposed that during the respiratory pause the asphyxial condition of the blood excited the bulbar centres, and produced a rise of blood pressure and stimulated the respiratory centre to action. Owing to the asphyxial spasm of the bulbar vessels the centres were but slowly oxygenated, and therefore remained in action for some little time. Filehne was able to produce Cheyne Stokes breathing by gradually constricting and then releasing the arteries conveying blood to the brain.

Hybernating animals exhibit most perfectly Cheyne Stokes breathing. I have observed the same phenomenon in the sleeping crocodiles at the Zoological Gardens. Young children when asleep exhibit to a slight degree group respiration, and I have found in my own child that the pulse shows the existence of periodic variations in blood pressure. This peculiar form of respiration may perhaps depend on our ancestral pedigree. Mammals which live in the water periodically come to the surface and take groups of respirations; pathologically and in hybernation the bulbar centres may revert to that ancestral rhythm which was associated with a lower metabolism and a sluggish life. I append a tracing (Fig. 31) taken recently from a patient. The upper line is the plethys-

mographic record of the arm. The blood pressure rises, just as in experimental cases, during each pause, until a group of respirations is finally excited. The rise of arterial tension, which caused the

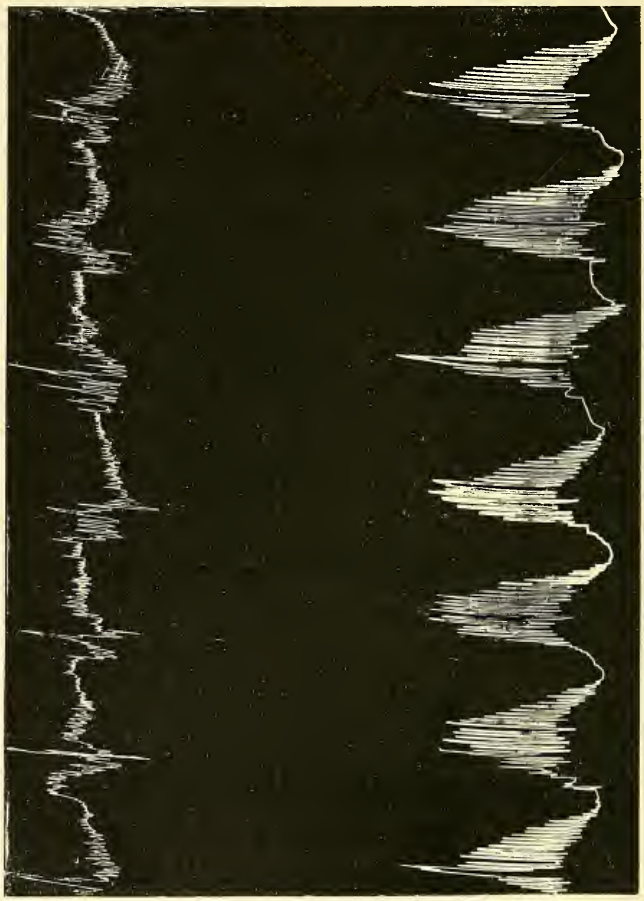


FIG. 31.

passive expansion of the arm in each pause, could be distinctly felt by the finger placed on the radial artery. Of these interesting phenomena I am engaged in making a special research, and I shall shortly, I believe, be able to establish

their uniform connection with conditions of cerebral anæmia.

A most interesting fact in regard to the symptoms of acute cerebral anæmia is, that the injection into the peripheral carotid under a high pressure of defribinated blood, produces to a minor degree exactly the same symptoms—namely, the slow pulse and rise of blood pressure. This has been obtained by Bergmann, François Franck, and Cybulski. The essential factor is the sharp application of the full pressure of the injecting fluid. Similarly, we have seen the Kussmaul and Tenner spasms can only be produced in rabbits by rapid ligature of the four arteries. If the ligatures be slowly applied some anastomosis is set up from the intercostal vessels and the spasms never occur, although the animals may die from respiratory or vaso-motor paralysis. I shall show that the same anæmic symptoms of slow heart and rise of blood pressure occur on suddenly increasing the intracranial pressure. This experimental result has been quoted as antagonistic to the anæmic origin of these compression symptoms, for it is said the forcible injection sends not less but more blood to the brain. I believe that this forcible injection does really succeed in producing anæmia of the medullary centres. The large arteries at the base of the brain are suddenly distended to the uttermost by the high tension of injection. A high arterial pressure minus the resistance of the arterial wall is transmitted directly to the brain substance, and is thrown on to the capillaries and veins and obliterates them (see Fig. 12). In a

short space of time the full arterial pressure reaches the capillaries through the arterioles, and the temporary anæmia then ceases.

We must remember "two sets of vessels are arranged in the pia mater side by side, or interlaced in a delicate network The one set feeds, the other drains, the capillaries." Now if the cerebral arteries *suddenly* expand beyond a certain limit, a process of *temporary* self-strangulation of these vessels takes place. The circulation itself for a short time stops, and the symptoms of acute cerebral anæmia are produced. Since the capacity of the bulbar vascular system is far less than the cerebral, and since the expansibility of any one bulbar vessel is no greater than that of a cerebral vessel of equal diameter, it follows that the total expansibility of the cerebral system far surpasses that of the bulb. Therefore, when blood is forcibly injected into the carotid, the cerebral system must expand at the expense of the bulbar system. The circulation again rights itself in these conditions as soon as the pressure in the occluded bulbar vessels rises to the static injection pressure. In the same way anæmic spasms may be produced by suddenly lowering intracranial pressure from a very high to a very low point. If the intracranial pressure be artificially raised to 100 mm. Hg and then suddenly lowered to 0, the cerebral arteries which were before compressed now expand to the utmost by that high blood pressure which exists in such conditions of intracranial pressure. By extension of the cerebral arteries the blood flow through the bulbar capillaries may be temporarily

stopped, for when the high intracranial tension is removed the blood-vessels suddenly change from rigid to expansile tubes. A greater volume of blood is needed to fill the expanded tubes, and therefore as the pump continues to deliver the same quantity per unit of time a temporary anæmia results; and this anæmia will be greatest in that part of the brain where the sum of the vascular expansion is least.

Having thus studied the immediate effects of the quadruple ligature, and the pathway of the anastomoses, I furthermore attempted to examine the remote effects. I ligatured with aseptic precautions all the four arteries in a dog. The wound healed throughout by first intention. The dog lived, and seemed for the first week after the operation to be exactly like Goltz's brainless dog—that is, a complete idiot. These are some of my notes on this animal:

*Experiment.*—Dog.

December 4, 1895 (3 P.M.).—Two carotids and vertebrals tied.

December 5.—The dog alternately sleeps and wanders round the room. When asleep powerful stimulations are necessary to arouse him. All his movements and tactile sensations are paresed. There is a continual tendency to turn in his walk, and generally to the left. He stumbles into every object. His pupils react to light, and the discs, when examined by Mr. Eve, appeared pale, with small arteries and large veins. Sounds are audible to it, as is evidenced by a pricking up of the ear. Smell seems to be the most efficient sense; he continually keeps his nose on

the ground, sniffing about as he wanders round the room. Whenever meat is put into his mouth he eats reflexly, but does not notice pieces of meat that drop or hang out between his teeth. He drinks reflexly when his nose is put into water. He showed no concern at a cat, although the cat spat with rage; nor at a lighted match, nor at tobacco-smoke blown into his face. He tries for many minutes to get through impossible objects—through crannies which are too small for him, bumps his head into every obstacle in the way, and finally drops asleep in front of such an obstacle. Sleep overtakes him at any moment during his perambulation. The scratching reflex did not seem to be exaggerated.

Mouth temperature equals 36° C.

Rectal temperature equals 39° C. Here was evidence of the deficiency of circulation in the head.

Pulse 120 high tension.

Respiration normal.

Urine passed normally.

December 6.—Much more susceptible to noise. Starts back on the firing of a match, and follows the light with his eyes like an infant. He shows, however, no fear of the light. There is still apparent no fear of a cat, for while the cat was hissing and trying to scratch, the dog calmly smelt round it. He showed difficulty in picking up and eating meat thrown before him. Walks more strongly.

December 7.—Came across the room when called, and seemed pleased at notice. Passed fæces.

December 8.—Answered by movements to a call,

and responds to patting. Seems frightened by a lighted match. Cannot localise a clip placed on his ear. Just as sleepy. Goes to sleep in any unnatural posture—on his back, or standing on his head, propped up against a cage. Quite uncertain how to jump down from a small height, gazes hesitatingly over the edge. Growls and turns to bite when irritated. All his acts appear reflex. Howled when meat was placed in his mouth, and then chewed and swallowed it.

December 9.—Much more obtuse. Would not eat or drink. Fed with a tube. Growls at being interfered with.

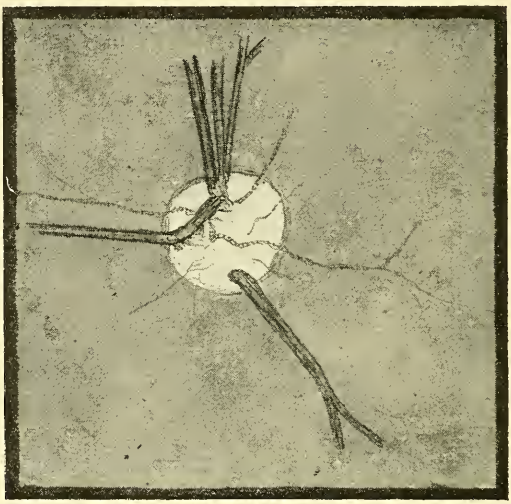
December 10.—Ate meat when placed in his mouth. Very little power of mastication. I injected glycerine into the rectum, and the dog ran round and round the room many times, then had a motion, and straightway fell asleep; scarcely feels and does not localise a clip placed on the ear. When a string was tied on his tail he wound himself round and round a post, and then tried to get under his own tail. After this date the dog rapidly recovered.

On December 14 he was shown to the Physiological Society, and seemed practically a normal dog, save for some weakness and hesitation in jumping from heights.

On December 16 the dog was killed, the ligatures verified. The anastomostic arteries were found to be the intercostals and ascending cervicals, and mainly the branches of the superior intercostals. These last had become dilated up to the size of the vertebrals and supplied the basilar arteries, and thence the whole brain.

On a second dog I performed the same experiment. This animal rapidly recovered without any marked period of idiocy. Some general weakness of the limbs and increased sexual desire were the chief symptoms noted. The dog was killed at the expiration of a month and the ligatures verified. In a third dog,

FIG. 32.



general paresis and anæsthesia were much more evident. On the third day after the operation the animal walked about, but on a wide base with the legs straddled out. Artery clips placed on the ear or toes remained unnoticed, or at most provoked slight general uneasiness. Lighted matches and tobacco-smoke evoked no sign of fear. The dog smelt and ate food and responded readily to calls. I give here a drawing (Fig. 32) of the left optic disc kindly taken for me by Dr. Sequeira and corroborated by both his and my own observation.

The veins are seen to be very large and the arteries exceedingly small. The disc was pale but not swollen. On killing the animal I injected the arteries with a gelatine carmine mass.

The medulla oblongata and the whole of the base of the brain appeared well injected by way of the intercostal, ascending cervical, and anterior spinal arteries. The injection did not reach the upper surface of the cerebrum or cerebellum, and thus the sensori-motor and visual areas were uninjected. The temporo-sphenoidal lobe was injected; and this is of interest, because the dog responded readily to a call, while visual and tactile sensations were followed by no response showing cognition. After these experiments and that carried out by Astley Cooper there is not the slightest doubt that dogs can recover perfectly from ligature of all the four cerebral arteries. I am now engaged in attempting to gain the same results in regard to monkeys, and have found a condition of torpor and paralysis produced by ligature of both carotids and one vertebral. This condition came on first some hours after the operation. The brain appears to be wound up as a clock, and can run for some time with a deficient blood supply. The pathological changes during the period of idiocy will be of extreme interest. The microscopical examinations have been undertaken by a far more experienced neurologist than myself (Dr. Mott), and will form the subject of a further research.

In reference to the effects of acute cerebral anæmia in man there is a mass of experimental results.

Simultaneous compression of both carotids in the hands of Columbus and many others has, we have seen, produced loss of consciousness. Norman Chevers, in 1845, collected five cases of obliteration of both carotids in man without ill effects.

Kussmaul and Tenner compressed both carotids in six men, with the result that the pupils widened, respiration deepened, dizziness and loss of consciousness followed. In two cases general spasms resulted, and all the phenomena of a slight epileptic fit.

Schiff, by compression of his own carotid, produced spasms on the opposite side of his body, preceded by a sense of numbness and formication. I myself have twice produced clonic spasms in myself by compression of one carotid. The first effect on applying the compression was a sensation in the eye on the same side; then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm, then down the leg. Finally, clonic spasms of the hand occurred, accompanied by an intense feeling of vertigo and alarm. Consciousness of the clonus was aroused only by the sensation of the hand striking the arm of the chair as it went into clonic spasm. The central motor discharge in the brain seemed to me to be accompanied by no consciousness. These effects of compression of one carotid vary in different men, no doubt, in relation to the freedom of the anastomosis in the circle of Willis.

Kussmaul and Tenner thought spasms could only be produced when the hind brain was rendered anæmic,

but we now know this to be erroneous. These spasms in man are in every respect the same as in Jacksonian epilepsy, or the epilepsy produced by tetanisation of the cortex in the sensori-motor area. On occlusion of one carotid, compensation by the circle of Willis is undoubtedly not immediately complete. Cramer, v. Schultén and Jolly have observed this experimentally, and Horsley and Spencer have seen the same thing in monkeys, for on ligaturing one carotid after exposure of the brain, the pia mater pales on the side of the ligature, and the cortex becomes far less excitable to the faradic current. In an hour or so the circulation and the excitability are restored. There is one famous pathological case on record of an obliteration practically complete, in the case of a man, of both carotids and vertebrals destroyed slowly and without ill effect, anastomosis being set up. A report of this case is given by Norman Chevers. From physiological experiments we can be assured that ligature of both carotids can be carried out without fatal results if done slowly and at intervals of time. The great principle of operation should be slow occlusion of the carotid by means of a screw clamp, so as to allow time for expansion of the other branches of the circle of Willis. If an hour were taken over the occlusion no ill effects, such as hemiplegia, would, I believe, ever result. Degenerated and inexpansile arteries do, of course, enormously increase the risk. If the four arteries were treated in this way at intervals of some weeks, I believe the whole of them might be safely tied in many men, and yet sufficient anastomosis be

set up. This is, of course, an operation outside the limits of surgery. In tying the common carotids the ligatures should, if possible, be applied in two successive operations. Before complete occlusion the arteries should be gradually compressed by a screw clamp, in order to allow time for the setting up of anastomosis.

### EPILEPSY.

Gowers writes: "It has been widely held that the morbid action in the brain is excited by arterial spasm causing cerebral anæmia, and also that the convulsions originate from the primary discharge of a convulsive centre in the medulla (Kussmaul and Tenner), but that the loss of consciousness is produced by vaso-motor spasm in the brain. In each theory it is assumed that the vaso-motor spasm is due to the sudden over-action of the vaso-motor centre in the medulla." He concludes that "the vaso-motor theory of epilepsy is unlike unneeded, unproved, and inadequate." From experimental results there is no doubt that sudden anæmia of the medulla oblongata can cause spasm of an asphyxial type, and that sudden occlusion of one carotid artery can in some men produce a march of epileptic spasm preceded by an aura, and exactly similar to the spasms aroused by cortical excitation. Sudden anæmia can therefore provoke epileptic spasm. On the other hand, in the case of epileptic spasm excited by cortical excitation or absinthe, the vaso-motor centre shares in the spasm. The blood pressure is thus raised and the flow of blood through the brain

accelerated. No evidence is obtainable of the constriction of the cerebral vessels. Every experimental method employed to detect vaso-constriction has failed, and therefore the cerebral vaso-motor spasm theory, both of epilepsy and of puerperal convulsion, must be withdrawn from pathology.

It is interesting to note that anæmia of the bulbar centres can excite a clonus of respiration and of blood pressure—namely, the Cheyne Stokes and Traube-Hering curves. Similarly, anæmia of the cortex can excite epileptic spasm. Certain poisons in the blood, such as morphia, can provoke Cheyne Stokes and Traube-Hering curves; absinthe excites enormous Traube-Hering curves. Similarly, sometimes morphia and always absinthe provoke cortical spasms. In conditions of kidney disease, Cheyne Stokes and Traube curves are sometimes manifested, and similarly the puerperal convulsions of cortical origin are attributed to uræmic conditions. In all these cases apparently the same change of central nutrition can provoke either bulbar or cortical spasm. Infantile convulsions are excited "by sensory irritation, especially of the alimentary canal, by morbid blood states in the onset of acute diseases, in states of general exhaustion produced by diarrhœa, or by intense paroxysmal cough." In the light of experiment, it is clear that in the state of exhaustion after diarrhœa, the vaso-motor mechanism is so weakened that compensation for gravity is abolished, and a condition similar to shock induced, in which cerebral anæmia is the chief factor. In paroxysmal cough the venous blood will be dammed

back, an asphyxial condition of the cerebral centres produced, and thus spasm excited.

As to the dependence of idiopathic epilepsy on cerebral anæmia, whether produced by poison or diminution of blood-flow, there is no experimental evidence. We must fall back on to the theory of explosive discharge or temporary failure of unstable nerve elements. This instability is apparently not *immediately* associated with any known change in cerebral nutrition. In the light of the histological researches of Ramon y Cajal, it is reasonable to suppose that the insulation between the dendrons or branches of the association nerve-cells is weakened in the epileptic, and hence the nervous discharge becomes diffuse.

In a recent paper, Ramon y Cajal suggests that insulation between one group of cell processes and another is brought about by means of the neuroglia cells. These cells are especially numerous in the molecular or association layer of the cortex, and are found either with expanded or contracted branches. When expanded these cells may, he thinks, act as an insulating material, and prevent the spread of nerve-currents.

## SLEEP.

Hughlings Jackson, by his observations on the retinal vessels, established the constant relationship between sleep and arterial anæmia. We have every reason to suppose that the retinal circulation is an accurate index of the cerebral circulation.

In conditions of anæsthesia and narcosis the loss of consciousness is constantly correlated with a fall of blood pressure. Thus chloroform lowers arterial tension and increases venous congestion; ether produces at first partial asphyxia and subsequently lowers arterial pressure; morphia lowers arterial tension by slowing the heart; chloral lowers arterial pressure. Durham observed that the pia mater became anæmic in dogs during the sleep that followed chloroform anæsthesia. When the animals were aroused the brain flushed with blood, and paled again as they sank once more into sleep. The same change in the waking and sleeping state has been recorded by Mosso. Kennedy observed, in a man whose brain was exposed, "marked congestion of the vessels" during sleep, "more particularly what seemed to be the veins, whilst all the vessels assumed a dark hue." When the patient awoke, he noticed "first an increase in the rapidity of the circulation, and then a change in the colour of the blood, and it sometimes seemed as if even new vessels had made their appearance." Kennedy's observation is not incompatible with, but corroborative of, Durham's experiments. With arterial anæmia there comes in natural sequence venous congestion of the brain, especially if the body be placed in the horizontal position and the skull be opened. In my dogs and monkey, with the cerebral arteries ligatured, sleep was the constant phenomenon, coupled with venous congestion of the optic disc; and there can be no doubt that sleep or lethargy is associated with cerebral anæmia.

The cerebral blood-flow depends on the vaso-motor

centre and the tonicity of the splanchnic area of blood-vessels. By means of the arteriometer, Dr. George Oliver has shown that the blood pressure falls in the state of weariness after exercise. If at the end of the day's work the vaso-motor centre is less able to maintain splanchnic constriction, then the cerebral circulation slackens. In the same way the blood-flow through the brain is diminished by post-prandial dilatation of the splanchnic area. When sleep is courted, all excitations from the external world, which arouse the vaso-motor centre to activity, are naturally avoided, and the body is placed in the warmth so as to promote vaso-dilatation of the cutaneous areas. By the horizontal position, coupled with the fall of arterial tension, the venous congestion of the brain is established.

A lethargic temperament is commonly associated with a patulous abdomen, piles and chronic constipation, and these are signs of a lack of tonicity of the splanchnic vessels, and therefore of a sluggish cerebral circulation. *The vaso-motor centre is the hub round which turns the wheel of a man's active mental life.*

In weariness and narcosis we must suppose that not only the tonicity of the vascular mechanism diminishes, but that the blocks in the nerve-tissue, which oppose the spread of nerve-currents, are increased. Ramon y Cajal has recently suggested that the neuroglia cells share in the production of sleep. These cells are especially numerous in the molecular layer of the cortex, and this layer may, on account of its anatomical structure be regarded as the association field of the

cortex. The neuroglia cells are found either in the contracted or expanded state. When expanded, Cajal thinks these cells may oppose an insulating material between the association nerve-cell processes, which interdigitate but never coalesce with one another. Thus the spread of excitation would be broken and mental activity cease. Duval supposed that the nerve-cell processes were drawn apart from one another in sleep by amœbiform contraction. This hypothesis has not even the anatomical support (Kölliker) which Cajal finds for his fanciful theory.

## SUMMARY.

(1) The symptoms of acute cerebral anæmia are produced by rapid and complete cessation of the cerebral circulation.

(2) The rapid cessation of the cerebral circulation can be caused by : A. Occlusion of the whole cerebral arterial supply by ligature, embolism, or compression. B. Occlusion of all the venous sinuses. C. By bleeding from a large artery. D. By sudden vaso-motor paralysis coupled with the effect of gravity.

(3) The usual sequence of symptoms of acute cerebral anæmia is : A. Loss of consciousness. B. Respiratory spasm. C. Slow heart and rise of blood pressure with cessation of respiration. D. Fall of blood pressure, acceleration of heart, and death.

These symptoms are exactly comparable with those produced by asphyxia.

(4) The bulbar centres are therefore first excited and

then paralysed. The fatal symptoms only arise when the bulbar circulation ceases.

(5) In man, sudden anæmia of the cerebrum can produce Jacksonian epilepsy preceding the loss of consciousness.

(6) Vaso-motor paralysis may precede and cause respiratory paralysis, but usually respiratory paralysis precedes and causes the vaso-motor paralysis.

(7) Such poisons as chloroform or amyl nitrite on entering the circulation, by producing vaso-motor and cardiac paralysis, may cause symptoms exactly comparable to the paretic symptoms of cerebral anæmia. (Such poisons as chloroform or amyl nitrite when applied directly to the bulbar centres first excite and then paralyse.)

(8) When the animal is in a state of shock the excitatory stage of cerebral anæmia is absent.

(9) When the anæmia is slow in onset the excitatory symptoms fail to appear.

(10) Cheyne Stokes respiration and Traube-Hering blood pressure curves are very common in states of partial anæmia of the bulbar centres.

(11) In no animals examined does ligature of both common carotids and vertebral arteries entirely cut off the brain from blood.

(12) The anastomotic pathway that remains open is by way of the intercostal and ascending cervical arteries, which feed the anterior spinal and so the basilar arteries.

(13) In goats, horses, nearly all rabbits, and about forty per cent. of cats, this anastomosis is too slight to

maintain the activity of the bulbar centres. The efficiency of the anastomosis can be improved by gradual ligature of the four arteries.

(14) All dogs have survived the immediate effects of the quadruple ligature.

(15) Four dogs (Astley Cooper's and mine) have recovered completely from the quadruple ligature after an initial period of paralysis and idiocy.

(16) There is every reason to believe that both common carotids can be safely tied in men if, with intervals of time between, each artery be gradually occluded by means of a screw clamp so as to allow the expansion of the anastomotic pathways.

## SECTION VI.

### THE METABOLISM OF THE BRAIN.

From my experiments on cerebral anæmia it appears evident that the blood supply to the brain is normally far above its actual requirement. In order that life may continue, it is necessary that the brain should unceasingly obtain a certain quantity of blood. Ligation of all the cerebral arteries or veins immediately produces loss of consciousness and death from failure of the bulbar centres. In the struggle for existence the throat is exposed to the grip of the enemy, and the carotid arteries may thus be easily occluded. Nature has therefore provided the vertebral arteries as a second line of supply, and encasing these arteries in bone has protected them from violence. The anterior spinal, ascending cervical, and intercostal arteries form a third but inefficient line of supply. In relation to the question of the cerebral blood need, it seemed to me to be of great interest to determine the metabolism of the brain as measured by the exchange of blood gases. Mosso has recently brought forward experiments to prove that the brain is the seat of very active combustion. By a long series of thermometric observations he has reached the conclusion that the

temperature of the brain is frequently higher than that of the rectum, or even than that of the aortic blood. He has further found that the temperature of the brain rises still higher when that organ is stimulated to activity. Hitherto it has been impossible even by the most delicate thermometric instruments to demonstrate the formation of heat in nerve (Rolleston). The heat which is produced by the brain, therefore, must owe its origin, as Mosso himself suggests, to the activity of the nerve-cells. The position of Mosso, therefore, is that the thin cortical shell of the dog's brain is endowed with such active metabolism that it is capable of generating heat sufficient in amount to raise the brain above the temperature of the aortic blood which is ever streaming through its capillaries, and which is at the same time carrying away this heat from every part of the organ. It is extremely difficult to ascertain the true temperature of the blood. I found in a research with W. M. Bayliss that the sources of fallacy are very numerous. We ventured to negative the positive results obtained by Ludwig and Speiss on the formation of heat in the salivary glands, on the grounds that these authors did not obtain the true aortic temperature. In our experiments the temperature of the saliva never rose above the aortic temperature (*Journal of Physiology*, 1894). The cerebral circulation changes passively with every alteration of the general arterial or venous pressures, and is in direct relation to the latter. Claude Bernard and Heidenhain found the blood in the right side of the heart to be warmer than the aortic blood. Mosso's

results might be due to the fact that he did not obtain the true aortic temperature, or to the conditions of his experiments altering the relative distribution of the venous and arterial blood in the brain and the body. The methods employed by Mosso to excite cerebral activity, such as the injection of absinthe or strychnine, have an enormous effect on the cerebral circulation and on the relative distribution of the blood.

As Mosso's precautions against the above sources of fallacy seemed to me insufficient, I determined to investigate the gases in the blood of the carotid artery and of the cerebral veins. The research was carried out by D. N. Nabarro and myself. We collected simultaneously samples of blood from the carotid artery and torcular Herophili both in states of rest and in states of spasm excited by the injection of absinthe. The results were compared with samples obtained simultaneously from the carotid artery and the deep femoral vein. The blood gases were extracted by a simple and rapid gas-pump which I devised for the purpose. By means of this pump six samples of blood could be collected and analysed in the space of three hours; a small quantity of blood (10 c.c.) was sufficient to give results of accuracy; the working error of the pump was less than 1 per cent. (*Journal of Physiology*, 1894). The results of a long series of experiments showed us that the brain is not a seat of active combustion. The combustion of the muscles is vastly greater than that of the brain, both in rest and in spasm.

In the following table is a comparison of the average

difference between the blood gases in the torcular and carotid artery, and between those in the deep femoral vein and the carotid artery :

| | | REST. | TONIC FIT. | | | | |
|---|---|---|---|---|---|---|---|
| Carbonic acid . | Torcular | + 3·87 | + 4·06 | × | 3 | = | + 12·18 |
| | Femoral vein | + 8·76 | + 13·90 | × | 3 | = | + 41·70 |
| Oxygen . . | Torcular | − 3·42 | − 4·95 | × | 3 | = | − 14·85 |
| | Femoral vein | − 12·92 | − 13·75 | × | 3 | = | − 41·25 |

During the state of tonic spasm the blood-flow from the femoral vein and from the torcular is twice to six times as great as during rest. The actual results in the tonic stage must therefore be multiplied by some such average number as three or four.

From the above results it is clear that our experiments do not bear out the conclusions of Mosso. The brain in comparison with the muscle is not a seat of active combustion. This research explains how the activity of the brain can continue when the blood supply is very largely reduced.

## SECTION VII.

### CEREBRAL COMPRESSION.

The general symptoms of cerebral compression have been recorded from the earliest times. Galen noticed paralysis of movement and sensation following the careless introduction of a guard in the operation of trepanning. He recognised that the prognosis could be based more or less on the implication of respiration.

Boerhaave tells of a man at Paris who at times begged money in a piece of his own skull, his brain being only covered with the dura mater. "And he would frequently permit experiments to be made for a small trifle of money. Upon gently pressing the dura mater with one's finger, he suddenly perceived, as it were, a thousand sparks before his eyes, and upon pressing a little more forcibly his eyes lost all their sight; by pressing the hand still stronger on the dura mater, he fell down in a deep sleep, which was attended with all the symptoms of a slight apoplexy, merely by this pressure with the hand, which was no sooner removed but he as gradually recovered from the symptoms as they were brought on, the apoplectic symptoms first vanishing, then the lethargy, and lastly the blindness, all his senses recovering their former

perfection." Haller, in numberless experiments, found that on compression of the brain, dogs suffered pain, and on strong compression fell asleep and snored. La Péyronie recorded a most interesting case of abscess in the corpus callosum. The patient was plunged into a profound stupor. The abscess was opened and emptied, the man's senses then returned, only to vanish again as the abscess refilled. Injection of the sac with the syringe produced a like effect. The man could be thrown into stupor or awakened at will. The patient eventually recovered. La Péyronie, on the strength of this case, localised the soul in the corpus callosum. Friend, in his "History of Physic," speaking of Severus and other violent advocates of trephining, says "that whenever this operation has done good in the headache, vertigo, epilepsy, there has been either a foulness in the bone or pus, blood, or worms collected between the skull and the dura mater. It was a common custom among falconers to open the skull of their hawks in a vertigo with a cautery, from whence, say they, proceeds an ichor or sanious matter which, when it has done running, makes a perfect recovery." "In an apoplexy," he further writes, "we find that after all voluntary motions have stopped, respiration will still go on and the pulse beat. This does not happen thus because the heart and diaphragm receive their nerves from the cerebellum, but because they are muscles which have no antagonists, a less quantity of spirits being sufficient to carry on the vital functions though not capable of exerting the voluntary motions. And therefore we find wounds in the cerebrum are often cured, but in the

cerebellum seldom prove otherwise than fatal; and the symptoms which attend them give a pretty sure prognostic as vomiting, fainting, hiccough, and intermission of pulse." Somewhat later Magendie writes, "that if one presses the sac of a spina bifida while a hand is laid on the upper fontanel, one feels the brain expand as the sac empties. With greater pressure the functions of the nervous system are disturbed, the child falls asleep and into coma, shortly all the symptoms which occur when the volume of the cerebro-spinal fluid is artificially increased in animals." "Pressure on the belly," he adds, "can produce the same effect by driving back the venous blood." Later, Bergmann in the same way has put children to sleep and slowed the respiration by pressing the sac of a meningocele. Flourens wounded the arteries of the dura in young pigeons without opening the skull, showed that convulsions were produced by the extravasation of the blood, and these were relieved by opening the cranium.

The experimental study of the complete symptoms of compression is due to Leyden, Pagenstecher and Duret. Leyden drove albuminous fluid into the intracranial cavity of a lightly morphinised dog. As the pressure of injection rose the symptoms occurred in this order—cries, convulsions, irregular respiration, coma; then slow and strong heart-beats, dilatation of the pupils with nystagmus, followed later by acceleration of heart, cessation of respiration. The pain, Leyden attributed to the tension of the dura mater. An injection pressure of 100 to 200 mm. Hg produced these culminating symptoms. The heart was slowed

by an injection pressure of 50 mm. Hg. The slowing of the heart disappeared on section of the vagi; and final acceleration of the heart arose when the vagus centre became paralysed. The primary cause of death was failure of respiration. A dog which had been brought by Leyden into a state of coma and convulsion, lived and recovered without any after-symptoms so soon as the compression was removed. When negative pressure was applied to the intracranial cavity, Leyden produced no results, except the local rupture of blood-vessels. Thus he established once and for all the experimental symptoms of compression and the fluid pressure necessary to produce them.

Further experiments were needed to show the volume of a foreign body which it is needful to introduce within the cranium in order to produce compression. Malgaigne injected water into a rabbit, and found one-sixth of the total volume of brain had to be injected in order to produce the symptoms. He left out of consideration the possible absorption of the water from the intracranial cavity. Pagenstecher, taking a non-absorbable substance, injected wax, and found 2·9 to 6·5 per cent. of cranial content necessary to produce death, while from 0·03 to 0·17 per cent. produced the slight symptoms. Duret also injected wax. He found the diminution of intracranial space by 5 per cent. produced coma, by 8 per cent. produced death. When Duret injected fluid rapidly into the skull under a high pressure, he produced hæmorrhages in the neighbourhood of the fourth ventricle, while the aqueduct of Sylvius and the central canal of the cord became

dilated. The heart and respiration stopped, the symptoms were relieved by section of the occipito-atlantal ligament. Concussion of the head by blows produced the same lesions. Duret explained the production of these concussion lesions in the following way. The bone is pliable and elastic, the blow depresses the bone for a moment, and hence a wave of depression passes down the brain and produces hæmorrhages at the seats of resistance.

Bergmann also injected wax and obtained the same series of symptoms. These he sums up from his own work and that of Leyden, Pagenstecher, and Duret as follows :

(1) Pain due to tension of dura mater.

(2) Stupor, sopor, coma follow.

(3) Clonic spasms and sometimes roll and circous movements, but these only when high pressure is suddenly applied.

The clonic spasms are the same as Kussmaul and Tenner's anæmic spasms, which also can be produced only when the anæmia is sudden in origin. Bergmann found spasm resulted from sudden compression of a meningocele, coma from slow compression. In cases therefore of traumatic hæmorrhage, with gradual bleeding into the cranium, these spasms do not occur.

(4) Slow heart, but acceleration if the vagi be cut, and slow deep snoring respiration followed by gasps at long intervals, and finally a rapid heart. To produce death intracranial pressure must equal the carotid pressure.

(5) Vomiting, emptying of the bladder and rectum.

(6) Constriction of the pupils first on the compressed

side, followed by dilatation. Nystagmus also occurs in the early stages. Bergmann concluded from a single experiment that the brain does not transmit pressure equally in all directions. He fixed a water manometer in a hole in the parietal region, and made a second hole 25 mm. away from the first, not opening the dura mater in the case of the second hole. He then found the necessary weight equal to just raising the water in the manometer which was fixed in the first hole. The weight was placed on the dura mater in the second hole. He then closed the second hole and made a third hole 30 to 40 mm. further away, and again found the weight necessary to raise the water in the manometer. The required weight was in this case two or three times greater, therefore he concluded the brain does not convey pressure equally in all directions. Althan attributed this result of Bergmann to the solid consistency of the brain itself: Niemeyer laid stress on the falx and tentorium which divide the skull into three separate chambers. The whole of the symptoms of compression were attributed by Bergmann to cerebral anæmia. In one experiment he found that obliteration of one carotid lowered the cerebral venous pressure in the proportion of 1 to 0·7. On injecting some wax into the cranium he obtained first a sudden increase of cerebral venous pressure, and then a fall in the proportion of 1 to 0·6; this experiment proved that cerebral anæmia was produced by the injection. Cybulski more recently has injected saline solution into the cranium, and found that the well-known symptoms arose when the blood-flow began to cease in the

carotid—that is, when intracranial pressure rose to that of the carotid. This he proved by means of his instrument the photohæmatochometer. Naunyn and Schreiber injected saline solution either through a hole in the parietal region or through the occipito-atlantal membrane, convulsions were produced by a pressure of 80 to 100 mm. Hg. v. Schultén, by injection of saline into the cranium of rabbits, arrested respiration by a pressure of 140 mm. Hg. Franck produced compression by means of a bag placed at the end of a tube, screwed into the skull and connected with a pressure-bottle and manometer. On sudden application of high pressure he witnessed cessation of respiration and inhibition of the heart. These did not occur if the animal was curarised or had had too much chloroform. On no apparent grounds whatever, Franck attributed these effects to vaso-constriction of the cerebral vessels. The results are obviously the same as the anæmic effects of Kussmaul and Tenner, and are caused by *sudden* anæmia of a normal and *excitable* bulb. Goltz frequently noticed temporary arrest of the heart and respiration, following his use of a method of removing the cortex of the brain by means of a forcible stream of water. It is therefore manifest that every researcher on the subject of compression of the brain—Leyden, Pagenstecher, Duret, Bergmann, Naunyn and Schreiber, v. Schultén, &c.—have obtained the same series of symptoms. The pressure of the injection fluid necessary to produce the culminating symptoms has been found by all of them to be about that of the carotid pressure. In the case of the importation of a foreign

body, such as wax, the decrease of the intracranial cavity must be by 3 to 8 per cent. in order to produce death.

Spencer and Horsley recently have recorded and minutely analysed the symptoms of compression. They employed a method similar to that of Franck ; a rubber bag was attached to a steel tube, which was in its turn screwed into the skull, and thick rubber tubing connected this tube to a burette full of mercury. The general symptoms obtained by these authors were the same as by all previous workers. The necessary diminution of cranial content to produce them they found to be about 5 c.c. in the dog, a result which agrees very well with that obtained by Pagenstecher. Spencer and Horsley write concerning their experimental method : " If the distension of the bag were unimpeded, only a very small column of mercury was necessary to distend it on account of the elasticity of the rubber. Therefore, when a bag was introduced into the closed skull, the amount of force required for the mere distension was neglected as being so extremely small. Consequently it follows that the degree to which the mercury sank in the burette and the height of the level of the mercury above the surface of the brain, accurately represented respectively the volume of the distended bag and the degree of pressure employed." On looking up the tables of their experimental results I find, among others, the following records. In one dog, 4·5 c.c. was driven into the bag by a pressure of 500 mm. Hg. In another dog, 8 c.c. was driven in by a pressure of 400 mm. Hg. The blood pressure at the moment of injection is recorded in the first case as

being 82 mm. Hg, in the second 121 mm. Hg. This shows that the elasticity of the bag, which no doubt might be kinked by the operation of screwing it into the skull and the resistance of the dura, was not eliminated in their experiments. By Spencer and Horsley's experimental method the amount of diminution of the cranial content can only be shown and not the intracranial pressure. Spencer and Horsley noticed in many of their experiments the dependence of respiration on the blood pressure. If the respiration ceased, a rise of arterial tension was able to start them once more. After division of the vagi, a rise of arterial pressure was also seen to directly affect the heart. It quickened as the blood pressure rose and slowed as it fell. These authors introduced the bag into the fourth ventricle in the upper part, and found that a moderate distension of the bag slowed the heart and raised the blood pressure without affecting the respiration. Similarly, when the bag pressed upon the upper end of the cervical cord and moderate compression resulted there, respiration alone might be affected. I would draw particular attention to the fact that these authors found the injection of 0·6 to 1·5 c.c. in the bag when placed over the bulb was sufficient to arrest the heart and respiration: over the cerebellum, 2 to 4 c.c.; over the cerebrum, 6 to 8 c.c.

The effect of a localised foreign body on the temperature of the animal has been studied by Dean.

I now pass to my own methods of studying abnormal conditions of cerebral tension. I raise the intracranial pressure by:

(1) The injection of an inert fluid into the cranium.

(2) By the production of hæmorrhage in the cranium.

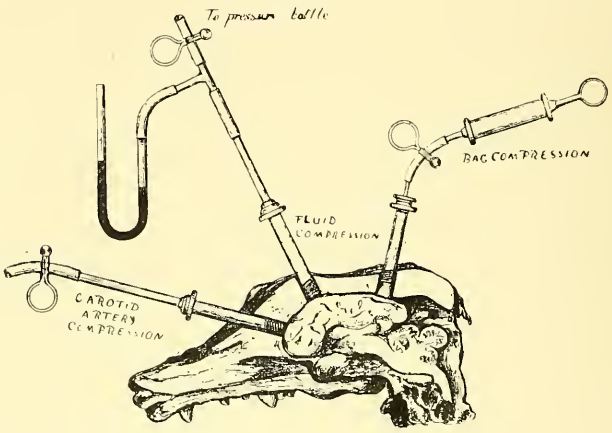
(3) By the local introduction of a foreign body of any given limited volume.

In the first case I proceed in the following way: I trephine the animal and screw into the skull a brass tube, and this is connected with a pressure-bottle and a mercurial manometer. Into the second trephine hole I screw the brain-pressure gauge. I record the arterial pressure by means of a mercurial manometer; the respiratory record I obtain by means of a Paul Bert or Marey tambour applied to the lower part of the thorax, or by a T-tube placed in the trachea. Connection in either case is made with a recording tambour. I register at the same time the pressure of the injecting fluid and the intracranial pressure. In some cases I measure the cerebral venous pressure in place of the intracranial pressure. In one class of experiments I have recorded intracranial pressure in the parietal region, in a second class in the cerebellar region, and in a third class over the upper end of the spinal cord. This last I effect by trephining the flat lamina of the atlas, opening the subarachnoid space and screwing in either a brain-pressure gauge or a brass tube connected with a manometer. The seat of injection has also been varied in the same way. Normal saline was the fluid generally injected. In other cases, either water or serum. Sometimes the fluid was warmed to the temperature of the body, at other times injected at the temperature of the room. The nature or the temperature

of the above fluids proved unimportant. The symptoms simply and solely arise from the pressure of injection.

In the second method I employ, my manner of proceeding is as follows. I connect the carotid artery to a brass injection-tube which is screwed into the cranium, and interpose a clamp. In the femoral artery I record the blood pressure. In all other respects the experimental method is the same. Blood is permitted to

FIG. 33.




flow from the carotid into the cranium, either into the subdural space or between the bone and the dura. The pressure in the femoral artery gives me the tension of the injection. In some experiments I have wounded a small vessel in the pia mater. The trephine hole has then been closed and the results recorded.

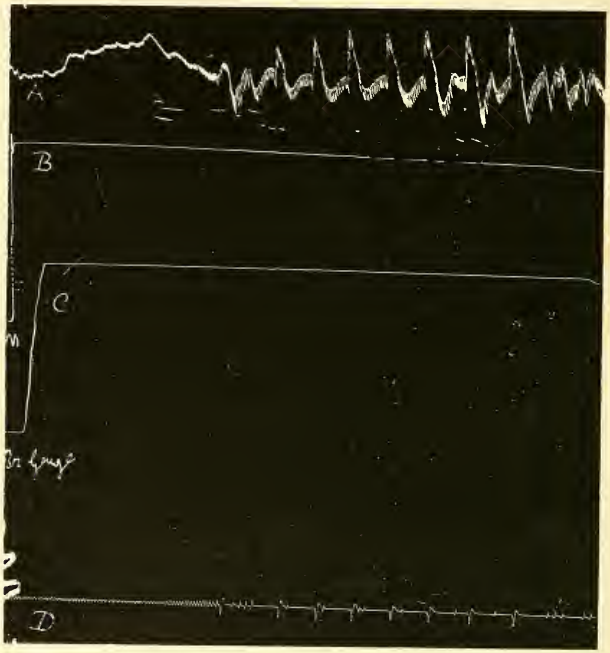
In my third method of investigation I proceed thus: A small india-rubber bag is attached to the end of a certain special piece of apparatus. This consists of an outer steel tube, which is screwed into a trephine hole, and an inner tube to which the bag is tied (Fig. 1, B).

This inner tube screws into the steel tube by means of a male and female screw. By means of a syringe the bag is distended at will with water, and the volume of the foreign body thus introduced is measured after each experiment by emptying the bag into a measure glass. The bag is distended either over the cerebellum or over the cerebrum. These three methods are shown in Fig. 33.

An important distinction must be drawn between compression by a foreign body of limited volume and compression by the continuous injection of fluid of a constant pressure. I will first consider the case of the continuous injection of fluid into the cranial cavity. If the fluid spread with ease to all parts of the cranial cavity, pressure throughout this cavity will then rise to the tension of the injection. The effect on the cerebral and spinal circulation is to cause all the veins and capillaries to become compressed, as the pressure of injection and the intracranial pressure rise, until when the injection pressure is made equal to the arterial pressure they are finally obliterated. Those capillaries and veins which are placed less favourably and offer greater resistance to blood-flow, must have a lower internal blood tension, and will therefore be obliterated by lower injection pressures. For the blood-flow will always follow the pathways of least resistance—that is to say, as the injection pressure rises, the circulation will be confined to the more open channels where the internal resistances are least. When the intracranial pressure reaches the arterial pressure, the cerebral circulation in these must also cease entirely. I have found, as all past

observers have, that when the tension of the injection fluid is raised gradually, fatal symptoms do not originate until the injection and consequently the intracranial pressure reach almost to the height of the arterial tension (Fig. 34).

FIG. 34.




A. Carotid artery. B. Pressure of saline injection.*
C. Brain-pressure gauge. D. Respiration.

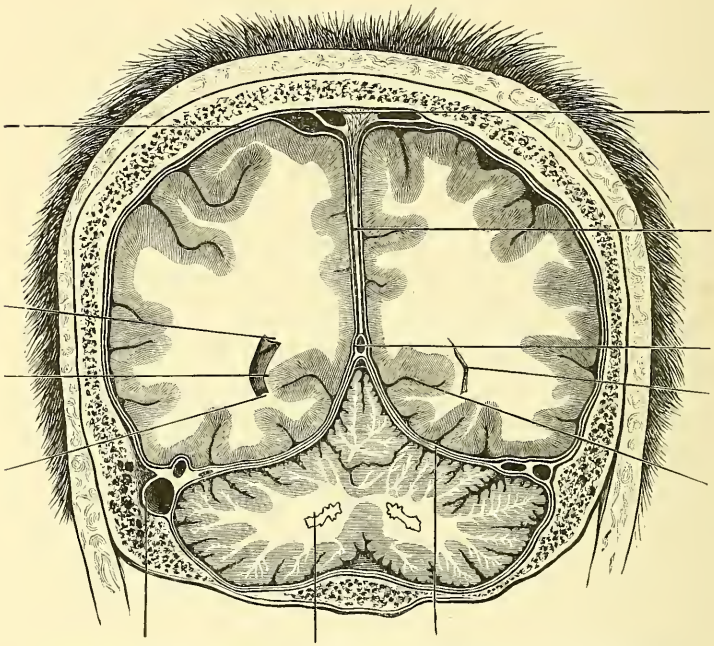
It will be remembered that, according to Poisseulles' law, as capillaries are diminished in diameter so blood-flow decreases by the fourth power. It is therefore evident that the bulbar centres can functionate with a greatly diminished blood supply. In reference to this point Nabarro and I have determined that the

* B and C are to be read in mm. Hg. The tracing is reduced one-third.

metabolism of the brain as measured by its output of carbonic acid is very low. If on injection the fluid does not spread easily to all parts of the cranio-vertebral cavity the distribution of pressure will then be different. I have evidenced that fluid can leak away from the cranial cavity with great ease. Now if there be existent considerable resistances to the spread of the injected fluid, and that fluid leaks from the cranial cavity, the pressure will then be greater at the seat of injection than at more distant parts. If the fluid does not spread at all, but forms a local collection at the seat of injection, the condition then becomes one of local compression. The blood will be expressed from the part of the brain which is compressed, until the local, cerebral, vascular tension rises to the pressure of the injection fluid. The brain, however, does not transmit an equal pressure in all directions when locally applied. This conclusion I have reached by simultaneous measurement in the cerebral, cerebellar and vertebral chambers. The brain can by no means be regarded as a bag of fluid enclosed in a rigid box; it is a viscous, inert mass of the consistency not of a bag of water, but rather of a lump of putty. The brain substance itself is incompressible, but the blood-vessels form an expressible sponge-work, lying chiefly on the outside of the brain mass. This brain mass can to a certain extent be dislodged from its normal position and moved in its entirety. On considering the anatomical relations of the brain, I find that each cerebral hemisphere lies in a separate chamber, partially protected from any increase of pressure in the opposite

hemisphere by the strong falciform ligament. The cerebellum and spinal bulb lie in a chamber protected from cerebral pressure by the tentorium cerebelli. This is a structure beautiful in contrivance in the form of an arch, supported by falciform ligaments above and

FIG. 35.



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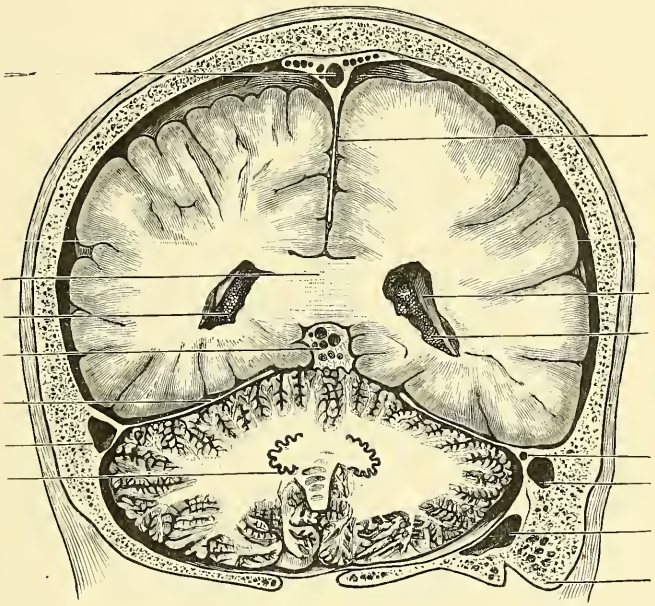
below (Fig. 35). The only connection between the cerebral and cerebellar chambers, is by the narrow isthmus tentorii cerebelli. This isthmus is accurately filled by the peduncles of the great brain (Fig. 36). The spinal cord lies in the vertebral canal, and this canal is connected with the cerebellar chamber by the narrow foramen magnum. The cerebellum and the bulb in their turn lie in close apposition to the margins of

the foramen magnum (Fig. 37). When fluid is rapidly injected into the subdural space in the parietal region, and under such a high pressure as 100 mm. Hg, I have found that the great brain is driven downwards against the base and completely blocks the isthmus

FIG. 36.



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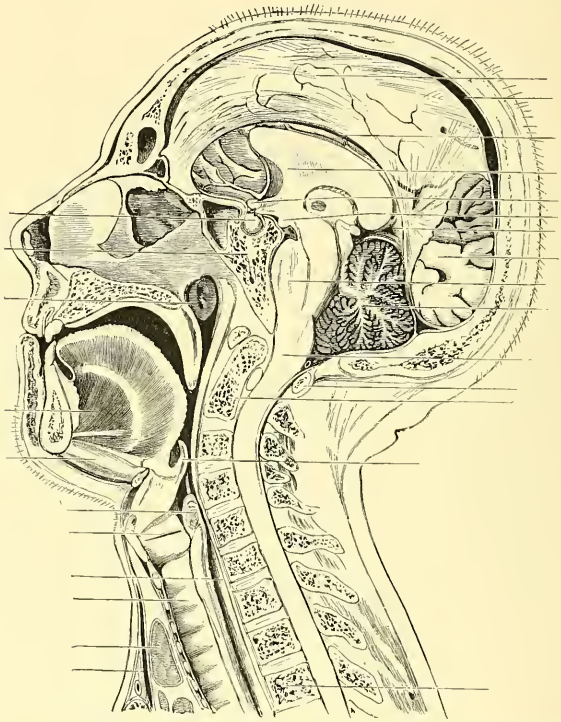
tentorii cerebelli, and at the same time the cerebellum and bulb descend and completely block the foramen magnum (Fig. 36). Thus, in many cases none of the injected fluid can leak into the vertebral canal. This fact can with great ease be experimentally proved. By merely opening the occipito-atlantal ligament and inspecting the effect of a parietal injection, the truth of this assertion will be granted. I must again insist on

the fact that the brain of the living animal fills the cranial cavity as closely as a finger in a glove. The normal amount of cerebro-spinal fluid is extremely small. It can be regarded merely as a lubricant for

FIG. 37.



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the brain in its pulsations. If, immediately after the death of a dog its cerebral hemispheres be removed from the cranium, the cavity can be filled to the brim with water and the head decapitated; there will then be discovered no leakage of water through the foramen magnum. This shows how accurately the cerebellar chamber fits the brain mass within it. When fluid is

injected into the cranium at low pressures the result is different. The fluid can now escape into the vertebral canal at each cerebral pulsation, for the cerebral vessels rhythmically decrease in size, and thus the fluid is enabled to find a way along the course of the blood-vessels. When, on the other hand, the injection pressure is high, this systole and diastole of the brain become impossible, for the blood-vessels cannot expand and become of the nature of rigid tubes. It has been apparent to me that the rate of escape of the injection fluid from one chamber to another varies in different individual animals, because it depends on the anatomical configuration of the brain, the depths of the sulci, and the relative volumes of the brain and cerebro-spinal fluid. The pathway of escape can be easily traced by injecting with a fluid coloured with methylene blue. In all cases of simple injections the fluid rapidly escapes altogether from the cranio-vertebral cavity by way of the veins. It can be seen issuing from the torcular Herophili. In order therefore to maintain pressure, the injection must be continuous. Directly the injection pressure-bottle is clipped off from the intracranial cavity, the brain-pressure gauge begins to show that the intracranial tension is rapidly falling to normal. I conclude, therefore, from the above considerations that the injection of fluid need not always produce an equal distribution of increase in intracranial tension. The passage of fluid from the cranial to the vertebral cavity either may not occur, or may not be rapid enough to compensate for the steady leakage which takes place by way of the cerebral veins.

That by the injection of a fluid local compression is sometimes produced, is proved by the fact that I have obtained the major symptoms of compression when the carotid pressure was roughly 150 mm. Hg with an injection pressure of 100 mm. Hg in the cerebral chamber, while a pressure of 50 mm. Hg has sufficed in the cerebellar chamber. The cause of these major symptoms is the cessation of blood-flow in the bulbar centres. To produce the anæmia of these centres any pressure above the capillary pressure, if applied locally to the centres, would be sufficient. The resistance in the bulbar capillaries need only be so far increased until that point is reached when the blood will flow through other pathways in the remaining parts of the brain offering less resistance. Now the bulb is so situated that fluid pressure cannot be locally applied to the bulbar centres: for example, if fluid be injected through the occipito-atlantal ligament, it will spread to all parts of the vertebral canal and will cause the base of the brain to float up. Thus, the tension in the cranio-vertebral cavity will everywhere rise to much the same point—that is, to the injection pressure. In the condition of things where all the vessels are equally compressed, there is no reason why any one capillary area should be obliterated earlier than any other, supposing, as we may, that the resistances to blood-flow in the given areas are equal. Therefore, the injection pressure must be raised almost to the arterial or static pressure before the major symptoms of apoplexy arise. Local compression can, however, be applied directly to the bulbar centres by the introduction of a foreign body

into the fourth ventricle, or, indirectly, through the descent of the brain mass as a whole, when a local pressure of sufficient height is applied to the cerebrum or the cerebellum. By the translocation of the greater or lesser brain, the bulb can be pressed against the bony margin of the foramen magnum, and the capillaries of the bulbar centres be thus obliterated. From these considerations it follows that the most unfavourable condition of compression is the local compression of the bulbar centres, and pressure applied there, and only there, but little above the capillary pressure, is sufficient to kill, for the blood then finds its way through the low resistance circuits of the greater and lesser brain, and avoids the high resistance circuit produced in the bulb. Local compression applied to the cerebellar cavity would cause fatal results so soon as it implicates the bulb, for the blood will in this case follow a low resistant circuit through the great brain. Local compression in the cerebral chamber is the most favourable condition, for then the circuit of low resistance lies in the cerebellar chamber, and the blood will flow through that so long as the pressure is not great enough to be transmitted to the cerebellar chamber.

I will now consider the effect resultant on hæmorrhage into the cranial cavity. Blood can form a local collection in the brain substance—in the ventricles of the brain, between the dura, and the bony wall of the skull, or in the subdural space. On the other hand, the blood can in some cases spread like an injection fluid to all parts of the cranio-vertebral cavity, entirely filling the subdural and the subarachnoid spaces.

Experimentally and pathologically both the localised and general distribution of the blood are to be found. In the case of the general distribution to all parts of the cranial cavity the cerebral capillaries will everywhere be uniformly compressed, and a pressure equal to the arterial pressure will be required to immediately produce complete anæmia and paralysis of the bulbar centres of life. The amount of compression of the cerebral vessels and consequent expression of blood can be exceedingly large before the bulbar centres are rendered completely anæmic. Thus, Bergmann records a case of meningeal hæmorrhage where, on post-mortem examination, a blood-clot was found weighing 180 grammes. In this patient the cerebral blood volume was therefore diminished by almost 180 c.c. before the bulbar centres became paralysed and life ceased. Experimentally, I have found that the introduction of blood into the cranial cavity produces symptoms which do not, as is the case with simple fluid injections, rapidly disappear when the pressure of the injection is withdrawn. The blood clots within the cranium and forms a foreign body, it cannot therefore leak away through the veins in the same way as an injected fluid such as normal saline or serum. On allowing blood to flow from the carotid artery into the subdural space the injection pressure is very high, equal to that of the arterial blood pressure; the blood therefore does not usually spread easily to all parts, but causes translocation of the brain mass while it remains itself localised, producing a local compression of that part of the brain where it is injected. Symptoms arise owing to the

translocation of the whole brain mass and the consequent pinching of the bulb against the bony wall of the cranium. The effect is, thus far, exactly the same as compression by a high-pressure fluid injection. As soon as the carotid artery is clipped off from the subdural space and the blood injection ceases the symptoms do not rapidly remit as in the case of fluid injections, but the intracranial tension remains raised.

This is because the fluid only and not the corpuscular parts of the blood can be resorbed, and because the filtration pathways into the veins become blocked with blood corpuscles. Slowly some resorption takes place, and slowly the blood leaks into the other chambers of the cranium and the local cerebral pressure falls. The local cerebral tension does not, however, return to the normal, because blood-clots form and remain within the cavity.

In the next tracing is seen the effect on the intracranial pressure of a carotid hæmorrhage and of symptoms produced thereby.

(Fig. 38, reduced one-third.)

A. Femoral artery. B. Brain-pressure gauge.
C. Respiration.

The cerebral pressure falls very slowly from resorption. Cerebral circulation ceases. This is shown by the effect on the venous pressure in the torcular Herophili. Some time after the cessation of the hæmorrhage the circulation begins once more in consequence of the absorption of plasma and the symptomatic rise of arterial pressure. This is shown by the torcular pressure.

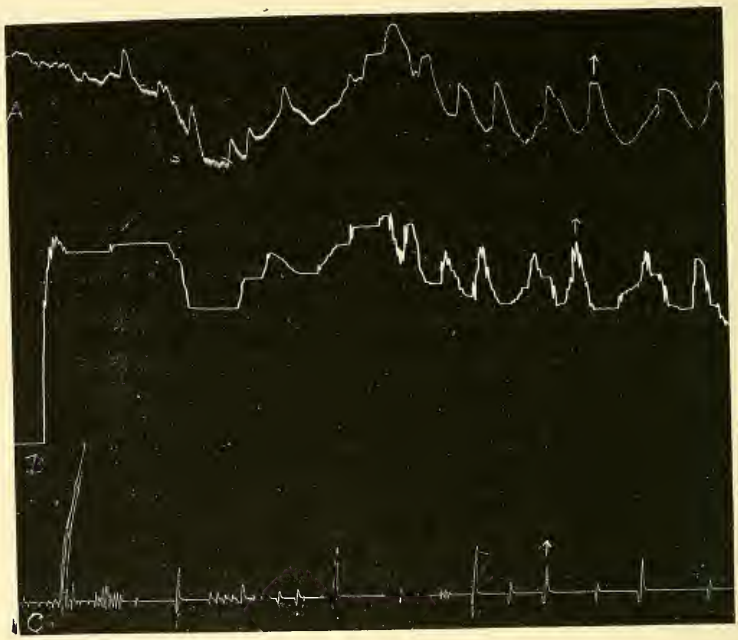
(Fig. 39, reduced one-third).

A. Femoral artery.

B. Torcular venous pressure.

The carotid artery was allowed to play into the cranium for a few seconds at the beginning of these

Fig. 38.



tracings. If the hæmorrhage and consequent clot be large, the remission of the symptoms does not occur after the cessation of the hæmorrhage. The remission can, however, be immediately obtained by freely opening the skull and clearing out the clot. It is very important to note that the mere opening of a small trephine hole is not sufficient to relieve the compression; the clot still

remains within, and the brain matter or clot presses up against the trephine hole and closes it up as a valve.

FIG. 39.

To entirely remove the compression the clot must either be cleared out or the hole must be made large enough to allow a compensatory expansion of the

volume of the cranial cavity. In this respect the experimental results are in exact agreement with surgical practice.

It cannot be too forcibly insisted that it is not the rise of intracranial pressure but the cerebral anæmia that produces the symptoms of apoplexy. When a blood-clot lies in the cranial cavity and all active hæmorrhage has ceased, the clot forms a foreign body which takes up a certain volume of the cranial capacity, and decreases to a corresponding extent the cerebral blood volume. For the amount of cerebro-spinal fluid that can be expressed from the cranial cavity is insignificant, and the brain matter itself is incompressible. It is the blood, therefore, that is expressed from the brain matter. This expression of blood is greatest at the seat of the clot and least at the distant parts of the brain. Intracranial pressure is that tension which remains after the force of the heart has been expended in driving the blood through the cerebral arterioles. When the cerebral capillaries and the vennules are generally narrowed, and finally obliterated in order to make room for a high-pressure fluid injection which distributes itself equally throughout the cranial cavity, then the blood pressure rises in the brain everywhere to the static or arterial pressure. In consequence the intracranial tension everywhere becomes raised to the arterial pressure. On the other hand, in the case of the injection of a localised foreign body the pressure will be raised to the arterial pressure in that part of the brain where the capillaries and veins are entirely obliterated by the injection. In the neighbouring part

of the brain where the vessels are diminished and not obliterated the pressure will be less, and in the most distant parts which lie in another chamber of the cranio-vertebral cavity pressure may even remain normal. Major symptoms of apoplexy will arise only when the bulbar capillaries are obliterated. This depends on the seat of the hæmorrhage and the direction in which the brain as a whole is pushed. Fatal results will ensue from a hæmorrhage in the cerebellar chamber far smaller than that needful in the cerebral chamber, for the pressure continuity between the two chambers is largely broken by the tentorium cerebelli. A very small local hæmorrhage in the neighbourhood of the bulbar centres will suffice to obliterate the bulbar capillaries and produce death.

By the injection of wax or the distension of a bag within the cranial cavity (as in my third method of experiment) a foreign body can be introduced exactly similar to a blood-clot. So soon as the clip is placed on the tube after the bag has been distended with the syringe, the bag, converted into an inert mass, takes up a certain amount of the cranial volume and maintains the expression of cerebral blood to a corresponding amount. The cerebral tension is locally raised to the arterial pressure, not that the bag any longer exerts pressure, but because by its presence the capillaries and veins are obliterated. This obliteration was initially produced by the pressure of the injection syringe. The permanent symptoms caused by the introduction of such a foreign body will entirely depend on its volume and locality, for, unlike the case of a simple injection

fluid, it is quite unabsorbable. In dealing with the effect the points which must be remembered are :

(1) That there is a pressure discontinuity between the chambers of the cranio-vertebral cavity.

(2) That the brain does not transmit pressure equally in all directions.

(3) That the bulbar centres must be compressed to produce the major symptoms of apoplexy.

If the bag be distended with two or three c.c. of water in the parietal region, and the brain tension be felt with the finger, or measured with the brain-pressure gauge in the opposite parietal region, in the cerebellar chamber, and in the vertebral cavity, it will then be found at once that the tension is very high in the parietal region, lower in the cerebellar region, and normal in the vertebral cavity. On inspection, the brain mass will be seen to descend into and block up the foramen magnum, and hence the pressure in the vertebral canal is unaffected. By this descent of the brain as a whole, the tip of the calamus scriptorius in the dog comes to lie in the vertebral canal, and thus the lowest bulbar centres may escape compression against the bony margin of the foramen magnum. In man the bulb lies, I think, at too high a level in the cranium for this to be possible. A certain very small quantity of cerebro-spinal fluid is expressed from the cranial into the vertebral cavity when the bag is first distended, and pressure is temporarily raised in the spinal subarachnoid space. The fluid is, however, quickly absorbed into the veins, and therefore the cerebro-spinal fluid can be almost entirely neglected in the considera-

tion of the production of cerebral compression. It is absolutely erroneous to teach that the increased cerebral pressure is transmitted to all parts of the cranio-vertebral cavity by the cerebro-spinal fluid. Adamkiewicz was quite right in saying that, "as every tendency of cerebro-spinal fluid to take a higher tension is paralysed by the passing of the fluid into the blood-vessels, and this resorption goes on until the tension is equal to the blood pressure, so the cerebro-spinal fluid cannot be the cause of cerebral circulation disturbances." He had, however, no justification in going on to assume that cerebral compression was impossible, and that a high intracranial pressure never existed pathologically. In his experiments with foreign bodies he never introduced a sufficient volume to produce symptoms, and contradicted the whole of clinical evidence with insufficient and incomplete experiment.

In the dog I have found, in confirmation of Spencer and Horsley, that the bag must be distended by 1·5 to 2 c.c. in the cerebral chamber in order to produce the first signs of the major symptoms, by 4 to 6 c.c. to cause complete bulbar anæmia and death. In the cerebellar chamber the bag must be distended by 1 to 2 c.c., and in the fourth ventricle by 0·5 to 1 c.c. to produce an immediate fatal effect. Nothing could demonstrate more clearly than these figures the fact that the brain does not transmit pressure in all directions, and that local obliteration of bulbar capillaries is all that is required to produce death. The symptoms of compression are, I believe, entirely due to cerebral anæmia.

There is no shadow of evidence to show that the bulbar centres can be mechanically excited by pressure. In the spasms of strychnine the intracranial pressure may rise to 50 mm. Hg in consequence of the rise in general blood pressure, but this does not produce apoplectic symptoms, and neither does it produce bulbar anæmia. The nerve centres, however, are pinched by a tension of 50 mm. Hg. On the other hand, I have found that the local application of 50 mm. Hg pressure to the cerebellum may produce fatal apoplectic symptoms in consequence of the compression of the bulbar capillaries. These experimental results conclusively prove that no mechanical excitation helps to cause the compression symptoms. On the other hand, the symptoms of compression are exactly comparable to those of cerebral anæmia. They pass through two stages—the first excitatory and the second paralytic. I have also noted that the excitatory stage is only obtained when the animal is in good condition and not in a state of shock, just as is the case with cerebral anæmia. Again, as the compression is more sudden and intense, so is the excitatory stage the more pronounced. Thus, by the sudden application of high pressure to the brain, by the translocation of this organ *en masse*, and by the thrusting of the bulb against the bone, is bulbar anæmia caused more completely and rapidly than by any other method. In consequence of this the excitatory symptoms may be even more marked than in the case of ligature of the four cerebral arteries in the rabbit. Epileptic fits may result. I have recorded a typical fit from sudden hæmorrhage in the cranial

cavity. Complete inhibition of the heart accompanied by spasm of respiration is the most striking excitatory symptom (Fig. 40).

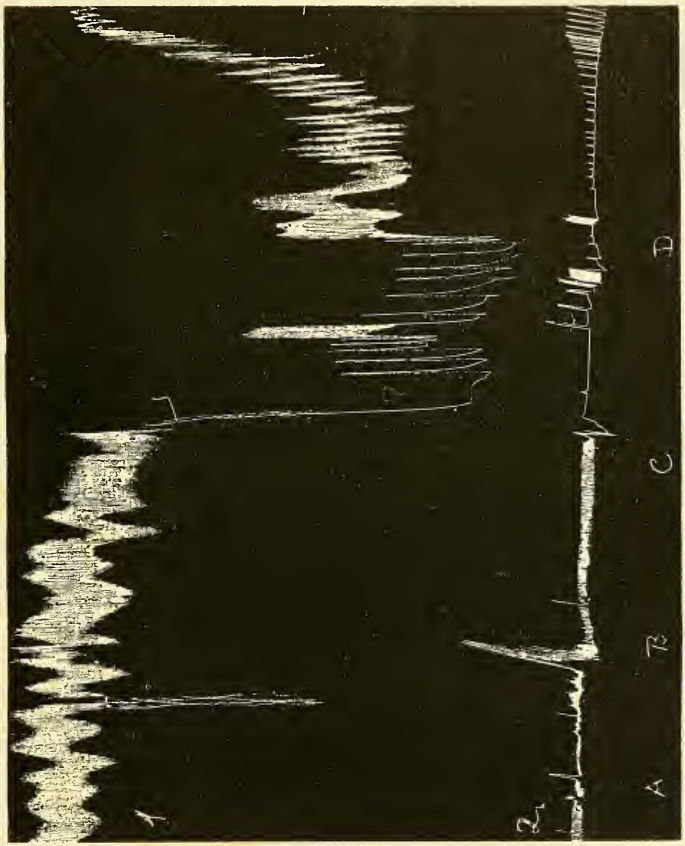


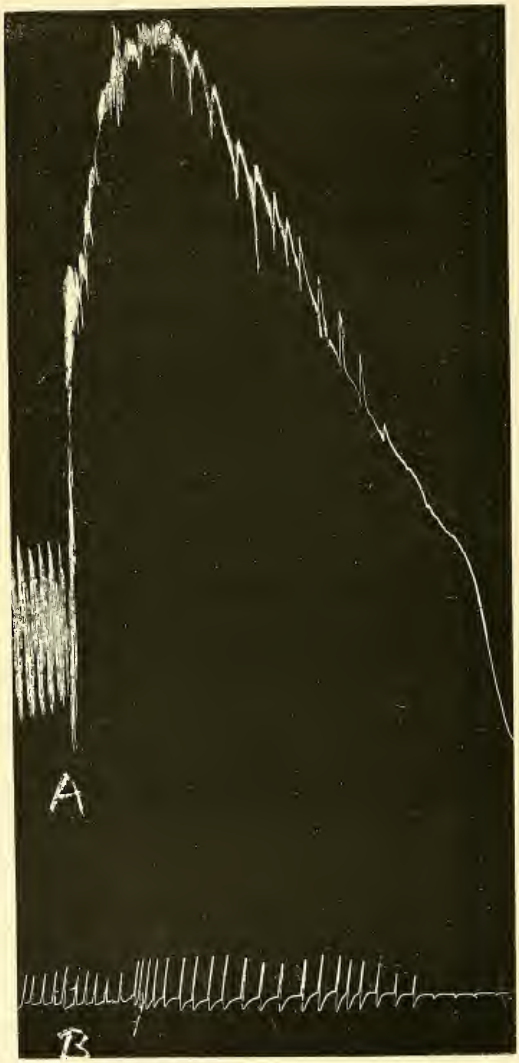

Fig. 40.

1. Carotid artery. 2. Respiration.
A. 2 c.c. in bag. B. 2 c.c. let out.
C. 4 c.c. in bag. D. 4 c.c. let out.

It is possible that the vagus nerve is excited directly by compression against the bone. When the vagus centre or nerve is paralysed or the vagi are cut, an

enormous rise of blood pressure becomes the most pronounced symptom. This rise of blood pressure

Fig. 41.




generally occurs if the injection pressure be withdrawn and then reapplied a second time (Fig. 41).

A. Carotid artery. B. Respiration.

4 c.c. in bag.

The vagus centre or nerve is exhausted by the first compression, and the vaso-motor centre reacts to the second. These striking excitatory symptoms, as shown in the tracings, are probably never caused by pathological hæmorrhage, because the compression is too slow at the onset. Moderately high blood pressure, slow and powerful heart-beats, and deep, slow, and laboured respiration form the excitatory symptoms of a gradually increasing compression.

In the second or paralytic stage the heart accelerates, the movements of respiration continue in the form of expiratory gasps, occurring at long intervals, and the blood pressure falls to zero. Traube-Hering blood-pressure curves and Cheyne Stokes respirations, as in the case of cerebral anæmia, are very frequent, especially during the period of recovery after the compressing agent has been withdrawn.

The causes of death in compression are similar to those in acute anæmia.

(1) Primary failure of respiration, with secondary failure of heart and vaso-motor mechanism.

(2) Primary failure of the vaso-motor centre with secondary failure of the respiration and heart.

The first sequence is the more common, the second often occurs when moderate compression is applied to an animal in a condition of shock, such as follows on the performance of previous compression experiments. The bulbar centres are then not rendered entirely

anæmic by the compressing agent, but become so in consequence of the failure of the exhausted vaso-motor centre. Compression of the abdomen in such cases is effectual in restoring the blood pressure and maintaining the respiration.

I will now turn to the consideration of the less immediate effects of an intracranial hæmorrhage. I will suppose that in a patient a localised meningeal or intracerebral or ventricular hæmorrhage has taken place, and that the effusion of blood has ceased. The patient will survive the immediate effects of the primary compression, except in those cases where the hæmorrhage has occurred in the locality of the bulbar centres. Such cerebro-spinal fluid as exists in the cranium and some of the effused plasma or serum will be absorbed, and thus the primary increase of cerebral tension lessened. Death may, however, occur secondarily from shock caused by the gradual and advancing paralysis of the vaso-motor centre induced by the primary compression. I have observed that this form of death occurs experimentally in some animals, even after an opening has been made in the cranial cavity and the compressing agent entirely removed. If, however, death from shock does not supervene the state of the patient will be as follows : There will be in the cranium a localised foreign body which occupies the space of a certain vascular area. and destroys the function of a certain portion of the brain. In consequence of the obliteration of the veins and capillaries, the local cerebral tension will be equal to that of the arteries which normally feed the affected area. In the oblite-

rated area there will be complete stasis of the blood. In the border areas the capillaries and veins will be more or less compressed in proportion as the increased local cerebral tension is transmitted to the brain substance. In consequence of this compression the blood pressure will be raised in these capillaries and veins and the blood-flow lessened. In the more and more distant areas of the brain the circulation will be more and more nearly normal, and the blood-flow may even be increased, because the blood takes the channels of least resistance. I believe that at this point a secondary increase of compression may be established in two ways. The opinions I shall set forth on this important subject are, however, still immature. The conditions are complex and extremely difficult to settle by experiment, and my researches on this matter are at present incomplete. The first of these mechanisms is that of congestive œdema. Bergmann has ably discussed this question of secondary œdema, but has attributed to the cerebro-spinal fluid a share in the production of compression which I believe to be impossible.

(1) The high blood pressure which exists in those capillary areas surrounding the seat of complete vascular obliteration will lead to increased transudation of fluid, since plasma may pass more easily into the brain substance than the blood through the compressed capillaries. The transudation will take place at almost arterial tension, will increase the volume of the foreign body, and so will lead to compression of other capillary areas. A circulus vitiosus is thus established and the cerebral

anæmia may spread indefinitely. The greater the area of capillary obliteration the greater is the danger of transudation surpassing resorption. The transudation of fluid into the meningeal spaces cannot increase the compression, for the fluid will pass away by the veins which are still patent. Transudation, on the other hand, of fluid from arterioles into an area of brain substance where the veins are compressed would increase the compression, for then transudation would take place faster than resorption, and œdema of the brain substance would arise. A hæmorrhage which obliterated the veins of Galen, and at the same time blocked the Sylvian aqueduct, would lead in the same way to an intraventricular transudation, and this would advance the compression to a fatal termination. Œdema may arise in another way. Suppose the surgeon has removed the compressing agent, the obliterated vessels now damaged by anæmia will once more fill with blood. Transudation of fluid and even extravasation of blood may then occur through the impaired vascular walls. This inflammatory œdema may lead to compression of other capillary areas. Dean has experimentally found that the brain areas, from which some little time previously a local compressing agent had been removed, contained 3 per cent. more water than normal brain areas. Therefore, secondary compression may arise after the primary compression has been relieved by trephining. The origin of hernia cerebri is no doubt to be traced to this inflammatory œdema. The trephine hole may not be sufficient to relieve the compression produced by this œdema,

because the brain presses up against the margin of the hole and closes it as a valve. I will now turn to the second way, in which I believe an increase of cerebral compression may arise. It is by the large rise of arterial pressure which is so often produced by a primary compression of a severe kind. Supposing a large hæmorrhage has taken place, the cranial cavity is filled with (1) blood-clot; (2) a compressed area of brain in which the circulation has ceased; (3) a more or less compressed area of brain in which the circulation still continues with difficulty. Now in such a condition, if arterial pressure rises, not only may the primary hæmorrhage be increased, but there is no room in the cranium for further expansion of the arteries. In and around the compressed area, if it be extensive, there lie a large number of arterioles in which the full static pressure exists, for the outlet from these vessels is blocked. The full effect of any increase of arterial tension will not only be felt in the arteries, but will reach these arterioles. The area in which static pressure exists will thus be greatly increased in the brain, and therefore any increase of arterial tension so far as it is transmitted directly through the brain substance, must increase the compression of other capillary and venous areas. *In physiological conditions the arterial extension of one vascular area is no greater than in another. On the other hand, in the pathological condition of compression the arterial extension of one vascular area has become greater than the rest.* The increase of arterial tension which is directly transmitted through the brain substance can never com-

pletely obliterate the capillaries or veins. This must be so, because arterial tension minus resistance of arterial wall is less than that full arterial tension to which the internal pressure in the capillaries and veins must rise so soon as they are obliterated. Any diminution of capillary diameter, however, leads to an enormous decrease in blood-flow, and the circulation may possibly become so sluggish thereby that anæmia may result. Œdema and capillary compression no doubt go hand in hand in furthering the mischief whenever the arterial tension rises.

I have tried to put this theory to an experimental test.

I have recorded the cerebral venous pressure in the torcular, the intracranial pressure and the arterial pressure, and have distended a bag 2–3 c.c. in volume in the parietal region of the intracranial cavity. The cerebral venous pressure first rose owing to the expulsion of blood, and then fell to the level of general venous pressure. The heart was strongly inhibited, as is usual in the excitatory stage of compression. The intracranial pressure in the cerebral chamber rose 120 mm. Hg. I then cut the vagi, the blood pressure immediately rose enormously from 140 to 270 mm. Hg. The cerebral venous pressure remained unaltered. It is clear, therefore, that this great rise of arterial pressure did not drive any blood through the brain into the torcular. This experiment does not decide, however, quite conclusively whether more or less blood reached the *bulb*, and I have as yet devised no experiment to settle this point. If the

four cerebral arteries be tied in the dog, and apoplectic symptoms be produced by the distension of a bag in the cranial cavity, then opening one carotid artery does not in any way diminish these symptoms.

The question is one of great importance, because, on the one hand, Falkenheim and Naunyn recommend that the blood pressure should be kept up by every means in conditions of cerebral compression; and, on the other hand, Bergmann, with whom I am on the whole in complete agreement, is inclined to the traditional treatment of lowering blood pressure by depletion. In the dog, the brain can be driven down by compression, until the tip of the fourth ventricle comes to lie below the foramen magnum, and so is thus partly cut off from the pressure. A rise of arterial tension may, and does, increase the blood supply to this part of the bulb in the dog. This is shown by the renewed excitation of respiratory spasm when the arterial tension rises. The fourth ventricle lies in man too far above the foramen magnum for this to happen so.

Depletion is only indicated when the arterial tension is high, and, as Gowers says, "the diagnosis of hæmorrhage should be reasonably certain;" since in thrombosis or acute cerebral anæmia, loss of blood will only do harm by weakening the cerebral circulation. Spencer and Horsley, on reasonable experimental grounds, have advocated compression of the common carotid as a physiological means of producing cessation of hæmorrhage. The immediate and free opening of the cranial cavity seems to me, in the light of

experiment, to be a proper treatment in many cases of hæmorrhage besides those of traumatic origin. When the blood pressure falls sufficiently, the blood is sure to coagulate and the hæmorrhage cease. Meanwhile, death from compression of the bulb is prevented, if the cranial opening be large enough to compensate for the foreign body, or if the extravasated blood and clót can be removed. Intracranial hæmorrhage might reasonably be treated on the same surgical lines as intracranial abscess, whenever death is imminent from compression.

Depletion of the brain can be carried out most physiologically by free purgation and determination of blood to the abdomen. Dilatation of other vascular areas by hot applications, mustard plasters and massage, also aid in the passive production of cerebral depletion. *Active constriction of the cerebral vessels cannot be brought about experimentally by cold or any other means.*

From the above considerations similar conclusions can be drawn as to the production of compression by other pathological conditions.

### TUMOUR OF THE BRAIN.

Gowers writes: "By the process of growth tumours destroy directly the adjacent nerve elements. In the infiltrating tumours, the morbid tissue elements grow between and enclose, and gradually destroy the nerve elements. In the non-infiltrating growths, the nerve elements perish before the compressing tumour, and the zone of softening around these growths is due to this destruction. In both cases, however, the

slower the process of growth, the less extensive and less complete the damage." "Intracranial tumours also exert distant pressure. A growth is a new mass within the skull which occupies more space than the tissue which it has destroyed, and so exerts pressure on all parts in that region of the skull. The nearer the parts are to the growth, the greater is the effect of pressure upon them. The more the pressure is limited in range by resisting structures, the greater is its immediate effect. The falx offers some resistance to the extension of pressure from one cerebral hemisphere to the other, but is often displaced by it. More effective is the resistance of the tentorium, and tumours in the small space beneath it may compress all the structures therein contained. Thus the pons is often considerably damaged by tumours of the cerebellum." This pathological doctrine is proved by experiment to be in most respects substantially true. I have shown that pressure is not equally transmitted to all parts of the cranial cavity. The brain can be locally compressed, the viscosity of the brain mass, the falciform ligaments, and the tentorium largely prevent the spread of the pressure to distant parts. The anatomical arrangement of the falx and tentorium tends to direct the pressure which is transmitted along certain definite lines.

In the cerebral chamber, the structures at the base of the brain, including the optic nerves, the veins of Galen and the Sylvian aqueduct, seem to be most exposed to the transmission of pressure. Distant

parts may be compressed by the translocation of the brain *en masse*, and this is more liable to occur when the application of the compressing agent is rapid. Thus, a large tumour at the top of the cerebrum may drive the brain downwards, and pinch the bulb against the occipital bone. It is important to remember that the rise of pressure is only circulatory in origin. The intracranial pressure of a patient with cerebral tumour would not be higher immediately after death than in any other corpse. Increase of tension is only produced by compression of capillaries. A tumour produces compression of capillaries by its growth. The cranial contents cannot be increased, and if the quantitative ratio of cell-tissue to blood-vessel is altered, it must be at the expense of the blood volume. Room for the new growth can only be found by compensatory expression of blood, or by atrophy of cell-tissues. If the tumour is slow in growth, brain atrophy may take place as fast as new growth, and in such a case intracranial tension need not be raised. It seems to me highly probable that a tumour of slow growth may attain considerable size without causing any rise of cerebral pressure. It is somewhat difficult to conceive how a tumour, unaccompanied by inflammation or hæmorrhage, can materially raise intracranial pressure. It can do so by an alteration of the ratio of cell mass to vascular supply, and by the growth of new arterioles and capillaries in which the internal resistances to blood-flow are less, and therefore the blood pressure higher than in other brain areas. The blood will then be

determined to the pathway of least resistance, and atrophy and capillary compression will take place in the surrounding brain areas.

Tumour can produce secondary and more acute attacks of compression by exciting inflammation accompanied by hæmorrhages, œdema of the brain substance, thrombosis of blood-vessels, and thickening of the meninges. Inflammatory products will then collect in the cranial cavity and diminish the blood volume by a corresponding amount. Internal hydrocephalus produced by the tumour blocking the aqueduct of Sylvius and the veins of Galen, will cause the greatest amount of compression and the highest rise of intracranial tension. Just as a very small foreign body is sufficient to destroy life when placed experimentally so as to produce local compression of the bulb, so in patients "a tumour in the fourth ventricle," according to Gowers, "frequently destroys life quickly before there has been time for much local lesion to be set up," and "a tumour of the same kind and size in the anterior part of the cerebral hemisphere may cause far slighter symptoms than if seated in the pons."

On experimental grounds the treatment for exacerbation of symptoms of compression in cases of tumour should be directed to lowering the blood pressure. The determination of the blood to the abdomen by free purgation is undoubtedly the most effectual method of effecting this.

In meningitis, tubercular meningitis and cerebral abscess, compression can arise by the accumulation of

inflammatory exudations, by inflammatory dilatation, by thrombosis and blocking of vascular areas; and whenever the Sylvian aqueduct and the veins of Galen are obliterated by intraventricular transudation. In all these pathological states a circulus vitiosus can be established leading to advancing cerebral anæmia. The pathological cause of ventricular hydrocephalus is no doubt to be found in blocking of the veins of Galen and the Sylvian aqueduct.

In meningocele there is no evidence of any compression symptoms except when the sac be compressed from without, for the origin of meningocele is traceable to malformations of the cranio-vertebral cavity, leading to distension of the wall by the normal intracranial tension. The spasms that arise sometimes after excision of a meningocele, probably owe their origin to compression. The relation of transudation to resorption must be altered by the extensibility of the cranio-vertebral cavity. When this space is suddenly and largely diminished by excision of the sac, the transudation of cerebro-spinal fluid may be more rapid than the resorption, and the cerebral circulation be unable to suddenly accommodate itself to the altered conditions.

In cases where increased amounts of cerebro-spinal fluid are found in the meningeal spaces, there is not the slightest experimental ground for supposing the existence of compression. Cerebro-spinal fluid in the closed cranium can only increase in proportion as the brain atrophies.

The operation for trephining for such cases as

general paralysis of the insane can receive no justification on any experimental grounds. "Serous apoplexy" is an impossibility; the increase of fluid is only a sign of passive venous congestion and cerebral anæmia. The cause of death in these cases, I have no doubt, is to be traced to the gradual cessation of the cerebral circulation produced by cardiac or vaso-motor paralysis, and determination of the blood to the splanchnic area.

## SUMMARY.

From the final section of my work I draw the following conclusions:

(1) The brain does not transmit pressure equally in all directions.

(2) The cerebro-spinal fluid cannot permanently transmit a local rise of pressure to other parts of the central nervous system. The fluid is rapidly resorbed from the cranio-vertebral cavity.

(3) There is a large amount of pressure discontinuity in regard to foreign bodies between the cerebral and cerebellar chambers. There is complete pressure discontinuity between the cranial and vertebral cavities.

(4) This pressure discontinuity is effected by (1) the viscosity of the brain substance, (2) by the tentorium cerebelli and falciform ligaments, (3) by the plugging of the isthmus tentorii cerebelli and the foramen magnum by the translocation of the brain mass.

(5.) The major symptoms of compression are produced by the anæmia of the spinal bulb, and are absolutely

comparable to the symptoms of acute cerebral anæmia otherwise produced. A far smaller foreign body kills in the bulbar region and in the cerebellar chamber than in the cerebral chamber.

(6) It is not the mechanical pressure but the cessation of blood-flow that produces these symptoms.

(7) Any pathological increase of cerebral pressure is circulatory in origin, a foreign body within the cranial cavity obliterates veins and capillaries, and raises the cerebral tension at the seat of obliteration from capillary to arterial pressure.

(8) A small opening into the cranial cavity does not necessarily relieve compression. The foreign body must be removed or the opening be large enough to allow an equivalent compensatory expansion of the cranial contents. In the latter case the viscosity of the brain mass may prevent the compensatory expansion, and local compression will then continue.

(9) When the primary compression is extensive, the secondary increase of compression can be brought about by congestive or inflammatory œdema, and by a rise of arterial pressure. A circulus vitiosus is established.

(10) The circulus vitiosis can also be established in cases of embolism, meningitis, cerebral abscess, ventricular hydrocephalus and cerebral tumour.

(11) The danger of this circulus vitiosus increases in proportion to the size of the primary area of vascular obliteration.

# BIBLIOGRAPHY.

## BY THE AUTHOR.


Hill. On Intracranial Pressure. *Proc. Roy. Soc.*, London, 1894, lv. p. 52.

Hill and Moore. Effects of Compression of the Common Carotid Artery. *Brit. Med. Journ.*, London, vol. i. 1894, p. 962.

Hill and Bayliss. On Formation of Heat in Salivary Glands. *Journ. of Physiol.*, London, 1894, xvi. p. 351.

Hill. The Influence of Gravity on the Circulation. *Journ. of Physiol.*, London, 1895, xviii. p. 15.

Hill and Nabarro. On the Exchange of Blood-gases in Brain and Muscle. *Journ. of Physiol.*, 1895, xviii. p. 218.

Hill and Bayliss. On Intracranial Pressure and the Cerebral Circulation. *Journ. of Physiol.*, London, 1895, xviii. p. 334.

Hill. Gas-pump. 1894–95, xvii. p. 353.


## CEREBRO-SPINAL FLUID.


Adamkiewicz. *Sitzungsb. d. k. Akad. d. Wissensch*, Wien, 88, 3, pp. 11–98, 231–355.

Bochefontaine. Note sur le liquide Cephalo-rachidien, &c., *Soc. Biol. Mém.* (*Compt. Rend.*), Paris, 5, 1880, pp. 65–72; *Arch. de Physiol.*, Paris, 1879.

Dean. *Journ. of Pathol.*, London, i. 1892, p. 26.

Falkenheim and Naunyn. *Arch. f. Exper. Path. u. Pharm.*, 1887, xxii. p. 261.

Flourens. *Arch. génér. de Méd.*, 1831, xxv. p. 133; *Gaz. Méd. de Paris*, 1848.

Rech. Expér. sur fonct. du Syst. Nerveux, 2me édit., Paris, 1842.

Key and Retzius.* Stud. in der Anat. des Nerven-system, Stockholm, 1875.

Luschka. Die Adergeflechte des menschlichen Gehirns, Berlin, 1855; *Zeitschr. f. ration. Med.*, 1859, vii. pp. 68–71.

Magendie. *Rech. physiol. et cliniq. sur le liquide céphalo-rachidien*, Paris, 1842.

Quincke. *Arch. f. Anat. u. Physiol.*, Du Bois-Reymond, 1872, p. 153.

Reiner and Schnitzler. *Centralbl. f. Physiol. Leipzig u. Wien*, 1894, 8, p. 684.

Schwalbe. *Arch. f. mikroscop. Anat.*, 1870, vi. pp. 1 and 261; *Centralbl. f. Med. Wiss.*, 1869, No. 30, p. 465; Lehrbuch d. Neurologie, Erlangen, 1881, pp. 775–792.

Vieussens. Neurographia universalis, Francofurti, 1690.

## CEREBRAL PULSATION AND CEREBRAL CIRCULATION.

Abercrombie. *Edinburgh Med. Surg. Journ.*, Nov. 1818, 14, p. 553; Path. and Pract. Researches on Diseases of Brain, &c., 1828.

Althan. Der Kreislauf in der Schädel-Rückgratshöhle, Dorpat, 1871.

Bergmann. *Deutsche Chirurgie*, 30, p. 274 *seq.*

Bernard. Leçons sur la physiol. du système nerveux, Paris, 1858. 2, p. 493.

Burrows. On Disorders of the Cerebral Circulation, London, 1846

Cappie. The Intracranial Circulation, Edinburgh, 1890.

Cash and Dunstan. *Proc. Roy. Soc. London*, 1891, p. 314.

Cavazzani. *Arch. ital. de Biol.*, Turin, 16, p. 23, and 19, p. 214; *Centralbl. f. klin. Med.*, Bonn, 14, p. 335; *Centralbl f. Physiol.*, Leipzig u. Wien, 1895.

Cramer. Exper. untersuch. u. d. Blutdruck im Gehirn, Dorpat, 1873.

Dean. *Journ. of Pathology*, London, i. p. 26. 1892.

Donders and Berlin. *Nederl. Lancet.*, March, April, 1850; Schmidt's *Jahrbuch*, p. 16, Bd. 69, 1851; Onderzœkingen ged. in het. phys. lab. Utrecht, 1850.

* Key and Retzius' Monograph contains a complete history of the classical researches on this subject.

Ecker. Physiol. Unters. über die Bewegungen des Gehirns, Stuttgart, 1843.

Falkenheim and Naunyn. *Arch. f. Exper. Pathol. u. Pharm.*, Leipzig, 1887, 22, p. 261.

Ferrari. *Wien Med. Jahrb.*, 1888, pp. 81.

Franck, François. *Journ. de l'Anat. de Robin*, 1887, pp. 267–307; Trav. du Lab. de M. Marey, 1877, pp. 273–292.

Fredericq. Trav. du Lab., i. pp. 9–54, 1886.

Gaertner and Wagner. *Wien Med. Wochenschr.*, pp. 602 and 639, 1887.

Gaskell and Shore. *Br. Med. Journ.*, London, 1893, i. pp. 105, 164, 222.

Geigel. Virchow's *Arch.*, Berlin, 1891, 123, p. 27, and 1890, 119, p. 93; Die Mechanik der Blutversorgung des Gehirns, Stuttgart, 1890.

Goujon. *Journ. de l'Anat. et de la Physiol.*, 1867, p. 106.

Grashey. Festschr. für L. A. Buchner, München, 1892, p. 61.

Haig. *Brain*, London, xvi. p. 230.

Haller. Elem. Physiol., Lib. x. vii. p. 301.

Heubner. Die luetische Erkrankung der Hirnarterien, Leipzig, 1874.

Hürthle. *Arch. f. d. ges. Physiol.*, Bonn, xliv. p. 582 *seq.*, 1889.

Jolly and Riegel. Virchow's *Arch.*, 52, p. 218, 1871; Untersuch. u. d. Gehirndruck, Würzburg, 1871.

Kellie. *Trans. Med. Chir. Soc. Edinburgh*, i. pp. 84 and 123, 1824.

Key and Retzius. Stud. in der Anat. des Nervensystems, Stockholm, 1875.

Knoll. *Sitzungsb. d. kais. Akad. d. Wiss.*, math.-nat. Cl., 93, 3, pp. 227–258, 1886.

Levy, A. G. *Proc. Roy. Soc. London*, 55, p. 275, 1894.

Lewy. *Arch. f. pathol. Anat.*, Virchow, 122, p. 146, 1890.

Longet. Traité de Physiol., 1869.

Lorry. *Mém. de math. et phys. Akad. roy. des. Sci.* (sav. étrang.), iii. p. 308, 1760.

Monro. Obs. on the Struct. and Funct. of the Nerv. Syst., Edinburgh, 1783.

Mosso. De l'Impartielle, xii. 1872; *Centralbl. f. d. Med. Wissensch.*, Berlin, 1872; Ueber d. Krieslauf im Gehirn, Leipzig, 1881.

De la Mure. *Hist. de l'Acad. roy. des Sci. Paris*, lxii. p. 33, 1749.

Nasse. Congestion zum Gehirn, Bonn, 1836.

Naunyn and Schreiber. *Arch. f. exper. Path. u. Pharm.*, xiv. p. 1, 1881.

Nothnagel. Virchow's *Archiv*, 40, p. 203, 1867.

Oliver and Schäfer. *Journ. of Physiology*, London, 1895, xviii. 230.

Oudin. *Rev. mensuelle*, No. 12, p. 923, 1877.

Ramon y Cajal. *Arch. f. Physiol.*, Du Bois-Reymond, 1895, p. 375.

Reid, John. *Edinburgh Monthly Journ. Med. Sci.*, 1846.

Reiner and Schnitzler. *Centralbl. f. Physiol*, Leipzig u. Wien, 1894.

Roy and Sherrington. *Journ. of Physiol.*, London, xi. p. 85, 1890.

Salathé. Trav. du Labor. de Marey, 1876.

Schlichting. *Mém. de math. et phys. Akad. roy. des Sci.* (savants étrang.), Paris, 1750, i. p. 185.

V. Schultén. *Arch. f. Ophthal.*, Leipzig, xxx. 4, p. 89, 1884 ; *Arch. f. klin. Chir.*, Berlin, xxxii. 1885.

Schultz. *St. Petersb. Med. Zeitschr.*, 1866, xi.

Stelling. Nervus Depressor, Dissert., Dorpat, 1868.

Symington. *Brit. Med. Journ.*, London, 1882.

Vulpian. Leçons sur l'appareil vaso-moteur, Paris, 1875, 1, p. 109, and 2, p. 122.

Wertheimer. *Arch. de Phys.*, Du Bois-Reymond (5), v. ii. p. 297.

## INFLUENCE OF GRAVITY ON THE CIRCULATION.

Basedow. *Wochenschr. f. d. ges. Heilk.*, Berlin, 1838.

Bond. *Brit. Med. Journ.*, 2, 1885, 1109.

Brissaud and Franck. Trav. du Labor. de M. Marey, 1877, 3, pp. 137–153.

Cybulski. *Med. Wochenschr. St. Petersb.*, 1878, No. 11.

Friedmann. *Med. Jahrb. d. ges. d. Aertze*, Wien, 1882, p. 197.

Goltz. *Arch. f. pathol. Anat.*, 29, p. 422, 1864.

Hales, Stephen. Statical Essays, Lond., 1733, ii. p. 33.

Hall, Marshall. *Med. Chir. Trans.*, 1832, p. 250.

Hermann, Blumberg and Wagner. *Arch. f. d. ges. Physiol.*, 37, p. 467, 1885, and 39, 1886, p. 371.

MacWilliam. *Brit. Med. Journ.*, 1890, 2, pp. 831, 890, 948.

Oliver, George. Pulse Gauging, London, 1895.

Piorry. *Arch. gén. de Med.*, 1826, 12, p. 527.

Regnard. Rech. sur la congestion cérébrale, thesis, Strasburg, 1868.

Roy and Adami. Waist Belts and Stays, *National Review*, Nov. 1888; *Brit. Med. Journ.*, London, 2, p. 1321, 1888.

Salathé. Trav. du Lab. de M. Marey, 2, 1876, pp. 345–401; and 1877, pp. 251–272.

Schapiro. *Jahrb. über Anat. u. Phys.*, 1881, ii. 60.

## CEREBRAL ANÆMIA.

Bastgen. Puls-frequenz u. Hirndruck bei capillarer Embolie der Hirngefasse, Würzburg Dissert., 1879.

Bell, John and Charles. Anatomy and Physiology of the Human Body, vol. ii. pp. 88–90.

Chevers, Norman. *London Med. Gaz.*, 1845, 36, p. 1140.

Cooper, Astley. *Guy's Hosp. Rep.*, London, vol. i. 1836, p. 457.

Corin. *Bull. d. l'Acad. roy. d. Sc. de Belg.*, Brux., 3 Sér. 14.

Couty. *Arch. de Biol.*, Paris, 1876; *Soc. Biol. Mém.* (*Compt. Rend.*), Paris, 3, 1887, pp. 296–298.

Duval. *Soc. Biol. Mém.* (*Compt. Rend.*), 1895.

Filehne and Kionka. *Arch. f. d. ges. Physiol.*, 1896.

Gad. *Arch. f. Physiol.*, Du Bois-Reymond, 1886, p. 543.

Gowers. Diseases of the Central Nervous System, 2nd edit., London.

Horsley and Spencer. *Brit. Med. Journ.*, vol. i. 1889, p. 457.

Kussmaul and Tenner. Molesch. Untersuch., 1857, Bd. iii. 1.

Markwald. *Zeitschr. für Biol.*, München, 1890, 26, p. 259.

Panum. *Zeitschr. f. klin. Med.*, v. Günsberg, vii.

Ramon y Cajal. *Arch. f. Physiol.*, Du Bois-Reymond, 1895, p. 375.

Schiff. Lehrbuch d. Physiol., 1858–59, p. 108.

Sokolow and Luchsinger. *Arch. f. d. ges. Physiol.*, 23, 283, 1880.

## THE METABOLISM OF THE BRAIN.

Bernard, Claude. Leçons sur la chaleur animale, p. 32, 1876.

Heidenhain, R. *Arch. f. d. ges. Physiol.*, iv. 1871, p. 558.

Mosso, A. *Proc. Roy. Soc. London*, 1892; Die Temperatur des Gehirns, Leipzig, 1894.

Rolleston. *Journ. of Physiol.*, xi. 208, 1890.

## CEREBRAL COMPRESSION.

Adamkiewicz. Die Lehre vom Hirndruck, &c.; *Sitzungsb. d. k. Akad. d. Wissensch.*, Wien, 88, 3, pp. 11–98 and 231–355, June, 1883; *Wien klin. Wochenschr.*, 1884, 8 and 9.

Althan. Der Krieslauf in der Schädel-Ruckgratshöhle, Dorpat, 1871.

Bergmann. *Deutsche Chirurgie, Kopfverletzungen*, vol. xxx. p. 274 *seq.*

Boerhaave. Institutes, 1742, vol. ii. p. 307.

Cybulski. *Centralbl. f. Physiol.*, Leipzig u. Wien, 1890, p. 834. *O Ucisku Mózgu*, Krakow, 1891.

Dean. *Journ. of Pathology*, London, i. 1892, p. 26.

Duret. *Arch. de Physiol.*, Paris, 1874; *Gaz. méd. de Paris*, 1877, Nos. 49, 50, 61; Étude expér. et cliniq. sur les Traumatismes Cérébraux, Paris, 1878.

Falkenheim and Naunyn. *Arch. f. Exper. Path. u. Pharm.*, Leipzig, 1887, xxii. p. 261.

Flourens. Rech. expér. sur fonct. du syst. nerveux, 2me édit., Paris, 1842; *Gaz. méd. de Paris*, 1848, No. 30.

Franck, François. *Journ. de l'Anat.*, de Robin, 1877, pp. 267–307; Trav. du Lab. de M. Marey, 1877, pp. 273–292.

Freind. History of Physic, 1727.

Horsley and Spencer. *Trans. Roy. Soc. London*, 1892.

Huguenin. Allgemeine Pathologie der Krankheiten des Nervensystems, Zurich, 1873.

Hutchinson. Lectures on Compression of the Brain, *Lond. Hosp. Rep.*, vol. iv.

Goltz. *Arch. f. d. ges. Physiol.*, 1876, p. 1 *seq.*

Leidesdorf and Stricker. *Vierteljahrsschrift für Psychiatrie*, 1867, p. 68.

Leyden. Virchow's *Arch.*, Berlin, 1866, 37, p. 519.

Naunyn and Schreiber, *Arch. f. exper. Path. u. Pharm.*, Leipzig, 14, p. 1, 1881.

Niemeyer. Handbuch der spec. Pathol. u. Therap., Berlin, 1871, ii. p. 168.

Pagenstecher. Exp. u. Stud. ü. Gehirndruck, Heidelberg, 1871.

De la Peyronie. *Mém. de l'Acad. Roy. des Sci.*, 1741.

V. Schultén. *Arch. f. Ophthal.*, Leipzig, xxx. 1884, p. 1–76; *Arch. f. klin. Chir.*, Berlin, xxxii. 1885,

# INDEX OF NAMES.


Abercrombie, 31
Ackermann, 37, 45
Adami, 101, 112
Adamkiewicz, 18
Althan, 8, 161
Baglivius, 6
Bastgen, 121
Bayliss, 40, 42, 45, 48, 62, 66, 73, 153
Bell, John, 117
Bergmann, 7, 8, 27, 71, 135, 158, 160, 161, 162, 176, 189, 193
Berlin, 7, 8
Bernard, Claude, 153
Bochefontaine, 26
Boerhaave, 156
Brissaud, 81
Burrows, 32, 33
Cash, 59
Cavazzani, 38, 45
Chevers, Norman, 142, 143
Columbus, 142
Cooper, Astley, 118, 121, 141, 151
Corin, 124
Cotugno, 26
Couty, 120
Cramer, 10, 45, 50, 55, 73, 143
Cybulski, 39, 72, 80, 135, 161
Dean, 14, 37, 53, 56, 164, 190
Dogiel, 46
Donders, 6, 7, 8, 35
Dunstan, 59
Duret, 7, 18, 158, 159, 160, 162
Durham, 147
Duval, 149
Ehrmann, 120
Engel, 33
Esher, 119
Eve, 137
Falkenheim, 14, 15, 18, 27, 37, 56, 71, 72, 193
Fallopius, 6
Ferrari, 120
Filehne, 131, 132, 133
Flourens, 158
Franck, 7, 8, 81, 135, 162, 163
Freind, 157
Gad, 130
Gaertner, 10, 38, 46, 57, 58, 64, 68
Galen, 5, 156
Gaskell, 59, 124
Geigel, 68
Gerdy, 104
Göltz, 137, 162
Gowers, 144, 193, 194, 197
Grashey, 2, 68, 71
Gulland, 45, 75
Hales, Stephen, 101
Hall, Marshall, 80, 108
Haller, 6, 26, 157
Halliburton, 23
Heidenhain, 153
Hermann, 81, 119
Horsley, 61, 143, 163, 164, 183, 193
Hürthle, 38, 46, 56, 59
Hyderabad Commission, 107, 109
Jackson, Hughlings, 146
Jolly, 7, 45, 46, 55, 119, 143
Kellie, 31 32, 33, 34, 67
Kennedy, 147
Key, 16, 17, 24, 71, 73
Kionka, 132
Koch, 71
Kolliker, 149

Kussmaul, 33, 35, 118, 119, 121, 135, 142, 144, 160, 162
Laurie, 107
Leyden, 6, 7, 71, 158, 159, 160, 162
Levy, 60, 61, 62
Longet, 6
Lorry, 6
Lower, 117
Ludwig, 48, 153
Luschka, 18
MacWilliam, 110
Magendie, 16, 17, 120, 158
Malgaigne, 159
Markwald, 121
Mayow, 6
Mendel, 81
Michel, 17
Monro, Alexander, 31, 67
Morgagni, 117, 118
Mosso, 7, 125, 147, 152, 153, 154, 155
Mott, 141
Mure, De la, 6
Nabarro, 68, 151
Naunyn, 14, 15, 18, 27, 37, 56, 71, 72, 162, 193
Niemeyer, 161
Nothnagel, 37, 45, 55
Oliver, George, 66, 105, 106, 148
Oudin, 7
Pacchionus, 6
Pagenstecher, 158, 159, 160, 162
Panum, 118
Pechlinus, 117
Péyronie, La, 157
Piorry, 78, 79, 80
Poisseuille, 120
Quincke, 17, 72
Ramon y Cajal, 146, 148, 149
Regnard, 111
Reid, 32
Reiner, 23
Retzius, 16, 17, 24, 71, 73
Ridley, 6
Riegel, 45
Rolleston, 153
Roy, 39, 40, 46, 53, 55, 57, 58, 59, 62, 63, 64, 73, 101, 112
Salathé, 7, 37, 80, 81, 104, 111
Sanderson, Burdon, 2
Schäfer, 66
Schiff, 142
Schnitzler, 23
Schreiber, 18, 162
Schuller, 37
Schultén, V., 36, 37, 46, 57, 143, 162
Schultz, 7, 45
Schwalbe, 17, 18
Severus, 157
Sequeira, 140
Shaw, Claye, 81, 93
Sherrington, 39, 40, 46, 53, 55, 57, 58, 59, 62, 63, 64, 73
Shore, 59, 124, 131
Speiss, 153
Spencer, 143, 163, 164, 183, 193
Starling, 48
Stelling, 46
Swieten, Van 117
Symmington, 13, 49
Tenner, 33, 35, 118, 119, 121, 135, 142, 144, 160, 162
Valsalva, 117
Vieussens, 18
Vulpian, 120
Wagner, 11, 38, 46, 57, 58, 64, 68, 81
Wertheimer, 40, 57, 64, 67
Willis, 6, 38

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